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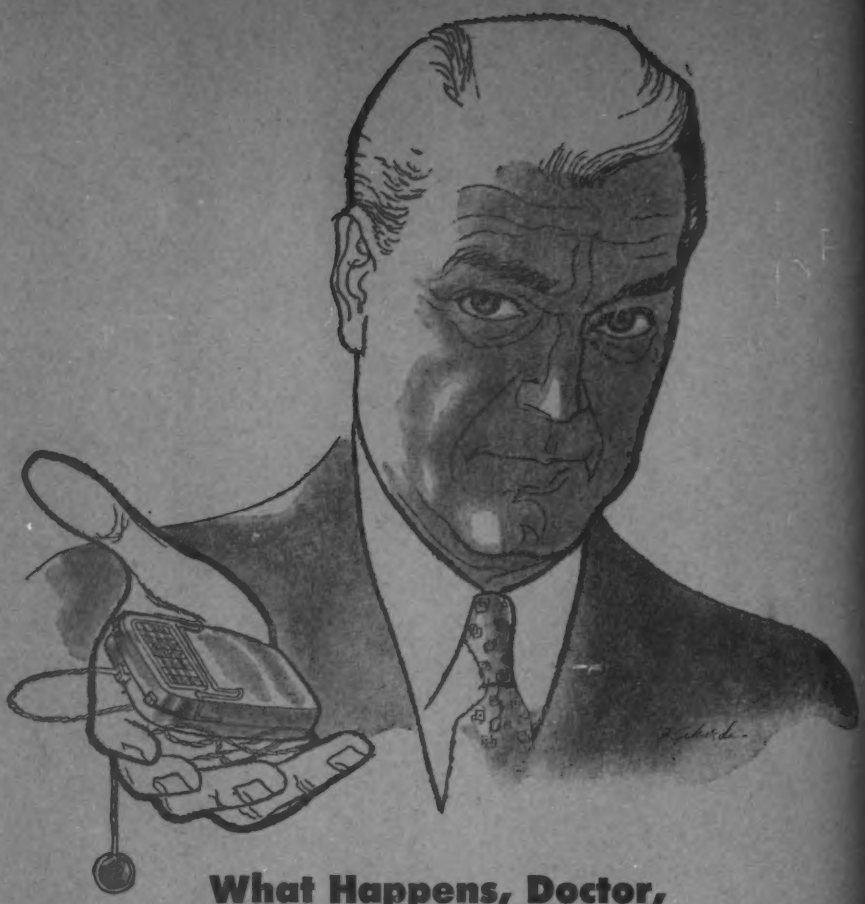

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THE
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VOL. LXIV JUNE, 1954. No. 6

THE CHEMICAL COMPOSITION OF PERILYMPH IN CATS.*†‡§

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There are few chapters in human physiology as rich in unsolved problems as that of the inner ear. A thorough knowledge of the basic physiology of the inner ear is obviously indispensable if we ever expect to answer the open questions related to otosclerosis, Meniere's disease, and a number of different types of deafness and vertigo.

This study was undertaken in order to establish certain basic facts on the chemical nature of the perilymph in cats and possibly to clarify the question of patency of the cochlear aqueduct.

REVIEW OF LITERATURE.

Review of the literature revealed rather meager information on the chemistry and physical properties of the labyrinthine fluids. The relative inaccessibility of the fluid spaces, the very small quantities of these fluids and the considerable technical difficulties in avoiding disturbance of normal physiology during experiments well explain the paucity of information on this subject.

Rossi[1] found the relative viscosity of the perilymph of the pigeon 1.7 as compared with 2.9 of the endolymph. Szasz[2] measured the refractive indices of the perilymph and cere-

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‡ This study was assisted by grants from the Research Council of the American Otological Society.

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brospinal fluid of the dog and concluded that the fluids were different.

Ledoux[3] found the refractive index of the perilymph in the cat and in the dog slightly higher than that of the cerebrospinal fluid. In addition, Ledoux noted that after perilymph was removed through the round window, the fluid was soon replaced and successive measurements revealed a progressive lowering of the index of refraction until the index almost reached that of the cerebrospinal fluid.

Aldred, Hallpike and Ledoux[4] and Ledoux[5] studied the osmotic pressure of the labyrinthine fluid of cats and found it higher than that of the cerebrospinal fluid; however, the osmotic pressures of the perilymph and endolymph were nearly the same.

The pH of the labyrinthine fluids in the cat was found to be higher than that of the cerebrospinal fluid (Ledoux[6]).

The chloride, urea N, and the reducing substance contents of these fluids were measured by Ledoux.[5] No significant differences were obtained.

Ledoux[5] mentioned that he found much more proteins in the perilymph than in the cerebrospinal fluid or endolymph of the cat.

One reference was found on living human material. Waltner and Raymond[7] analyzed perilymph from five patients who suffered from Meniere's disease. The protein concentration in perilymph was more than twice that found in the cerebrospinal fluid of the same individual patient. Analysis of endolymph of one patient showed similar quantitative relationship between endolymph and cerebrospinal fluid. The sodium contents of perilymph and cerebrospinal fluid were equal, according to these authors. They concluded that the identical concentration of cristalloids (sodium) and the great difference in protein concentration point was due to a semipermeable barrier between perilymph and cerebrospinal fluid.

As all human material originated from diseased labyrinths (Meniere's disease) it seemed important to obtain more data

from healthy animals, the anatomy of which was comparable to the human.

METHOD.

The middle ear of the cat was first exposed and blood, if any, was removed from the tympanic cavity, especially from the area of the round window. A very fine glass capillary was then passed through the round window membrane, and a sample of perilymph was removed with gentle suction. The bony overhang of the niche to the round window was not removed, in order to avoid any traumatism and hemorrhage in the immediate vicinity of the round window membrane. Perilymph was aspirated from both ears in each animal.

Samples of cerebrospinal fluid were obtained by cisternal puncture. The atlanto-occipital ligament was first exposed, and hemorrhage controlled in order to eliminate possibility of blood contamination.

A sample of the animal's blood was taken in each case for blood correction, in case microscopic amounts of blood were found in the perilymph, or in the labyrinthine cavities.

In every second animal cerebrospinal fluid was drawn first, immediately followed by removal of perilymph through the already exposed round window membrane. It was thought that this would reduce the possibility of mixing perilymph with cerebrospinal fluid via the cochlear aqueduct, by greatly diminishing the pressure of the cerebrospinal fluid in the subarachnoidal spaces.

The cat was decapitated on completion of the experiment, and the head was immersed in fixative fluid. The temporal bones and brain *in situ* were studied in serial sections, in order to ascertain possible damage to soft tissues. Tear of Reissner's membrane, or of the basilar membrane, was of special interest, because of the concomitant mixing of perilymph with endolymph. Serial sections were thought to be of importance in obtaining additional check on presence of hemorrhages of microscopic size in the labyrinth.

DESCRIPTION OF HISTOLOGICAL FINDINGS.

Thirty cats were used in these experiments, which were performed under general anesthesia.

Sixteen temporal bones were studied in serial sections. Those animals were selected for serial sections in which the fluids could be removed with minimal or no trauma to tissues, and in which the fluids obtained were macroscopically clear. Description of eight pairs of representative temporal bones is given below.

Group 1.—Histological findings of the ears, in which perilymph was removed first, followed by cisternal puncture.

Cat 814: Samples of perilymph and spinal fluid were macroscopically clear.

There was a clear-cut hole in the round window membrane on both sides. No tear of the membranous partitions of the labyrinth was found. There were a few red blood cells on inner aspect of the round window membrane of the left ear, while the right labyrinth was entirely devoid of free blood. Fibrin-like precipitate filled out most of the cochlear duct of the basal turn on both sides, while a small amount of similar precipitate was visible in some sections of the utricles and saccules. In several sections Reissner's membrane and the basilar membrane were found to be displaced in the direction of the scala tympani. The large vessel, which could be traced as leading to the stria vascularis, was bilaterally engorged especially in the basal turn.

Cat 815: Clear perilymph was obtained on the right side, while the left one was grossly blood-tinged (glass tip broke) and, therefore, discarded. The cerebrospinal fluid was clear.

Right ear: There was a small clear-cut hole in the round window membrane. No tear was found anywhere, but Reissner's membrane was found to be displaced downward in the direction of the basilar membrane in all turns of the cochlea. Few blood cells were present in the vicinity of the round window membrane. Considerable amount of blood was seen in the subarachnoidal spaces and in the cochlear aqueduct, none of which entered the scala tympani. Fibrin-like precipitate was present in the endolymphatic spaces only: cochlear duct, and saccule, utricle and ampullae.

Left ear: Extensive tear of the round window membrane and basilar membrane was found, in addition to a heavy hemorrhage in the scala tympani.

Cat 819: Both perilymphs and cerebrospinal fluids were clear. A small perforation was found in each round window membrane. The basilar membrane was found to be torn bilaterally as a result of sectioning. Reissner's membrane was depressed in all turns of the cochlea. Small amount of blood was seen in the scala tympani on both sides, more in the right one.

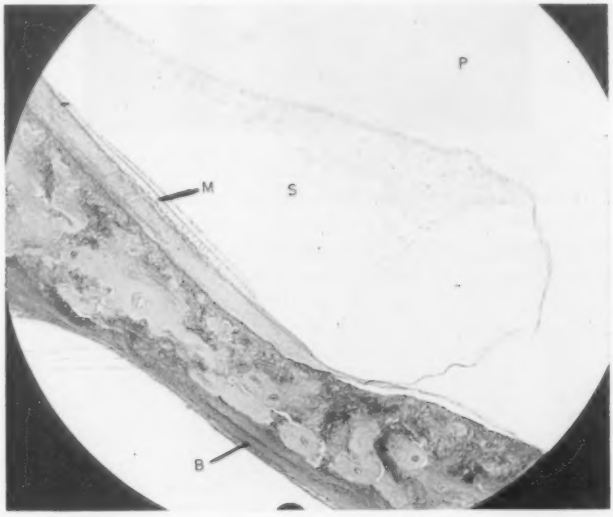

Fig. 1. Horizontal section through vestibule. S—saccule partially filled with precipitate; P—perilymphatic space, free of precipitate; M—macula of the saccule; B—bony labyrinth.

Cat 820: The perilymphatic fluids and spinal fluid were clear. A small opening was found in each round window membrane. These holes represented one-tenth of the entire window membrane. Fibrin-like precipitate was found in the endolymphatic spaces, mostly in the cochlear duct, some in the saccule (see Fig. 1) and utricle. Reissner's membrane was displaced downward in every turn of the cochlea. No blood

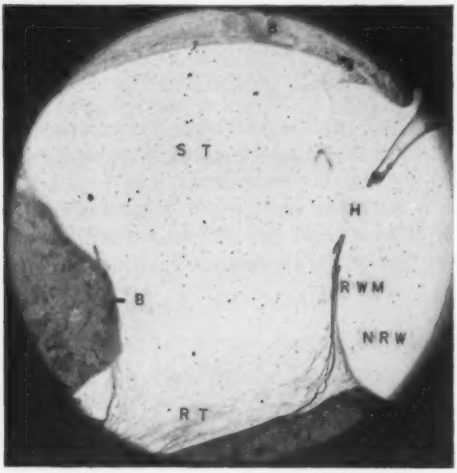

Fig. 2. Horizontal section through the round window membrane. H—perforation of round window membrane made by pipette; RWM—round window membrane; SC—scala tympani free of blood; NRW—niche to the round window free of blood; RT—reticular tissue of cochlear aqueduct; B—bony labyrinth.

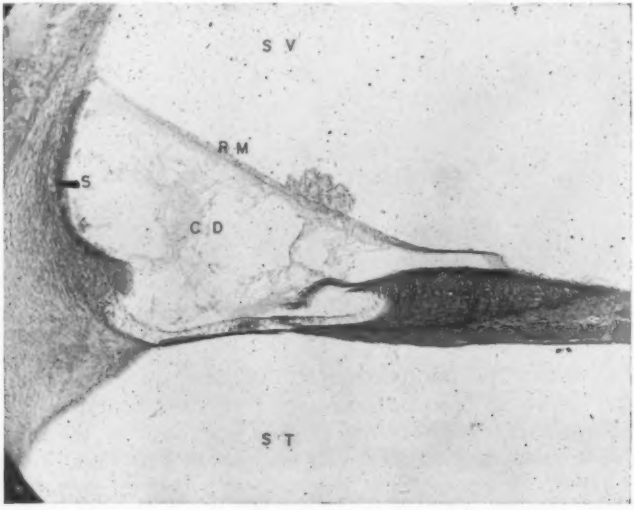

Fig. 3. Horizontal section through cochlea. CD—cochlear duct partially filled with precipitate; ST—scala tympani free of precipitate; SV—scala vestibuli, free of precipitate; RM—Reissner's membrane; S—stria vascularis.

was seen in the right labyrinth, while a few cells were present in the left one.

Cat 821: Perilymph specimens and spinal fluid were clear. Small, clear-cut openings were present in the round window membranes. There were a few blood cells near the opening into the left round window membrane. All membranous walls were intact, but Reissner's membrane and the basilar membrane were displaced in the direction of the scala tympani. The right labyrinth was entirely free of blood (see Fig. 2). Fibrin-like exudate was found in the cochlear duct of the first turn (see Fig. 3), in the saccule and utricle. Few blood cells were seen in the infravestibular portion of the left scala tympani.

Group 2.—Cerebrospinal fluid was removed first, followed by aspiration of perilymph.

Cat 822: Clear samples of perilymph and cerebrospinal fluid were obtained. A small hole was found in each round window membrane. The basilar membrane was torn in most turns, and the labyrinthine capsule was broken. Fibrin-like exudate was present in the cochlear duct. No trace of blood was found in the right labyrinth, while there were about 100 blood cells in the left scala tympani.

Cat 823: Clear fluid specimens were obtained. No tear was found in the membranous partitions. Blood, however, was seen in the scala tympani extending into the uppermost turn in the right cochlea. Very little blood was found in the scala tympani of the basal turn of the left cochlea. Fibrin-like precipitate was present in the cochlear duct.

Cat 824: Clear samples of perilymph, but somewhat cloudy cerebrospinal fluid were obtained. A small opening was found in each round window membrane. There were very few blood cells on the left side. No tear was seen on the right side, while the basilar membrane was found to be torn in several of the sections on the left. The labyrinthine capsule was broken on the left; the tear of the basilar membrane, therefore, was considered artefact. Fibrin-like precipitate was present in the cochlear duct and the saccule on both sides. A very small number of erythrocytes were scattered in this

fibrinous fluid. The vessel leading to the stria vascularis was greatly engorged (see Fig. 4).

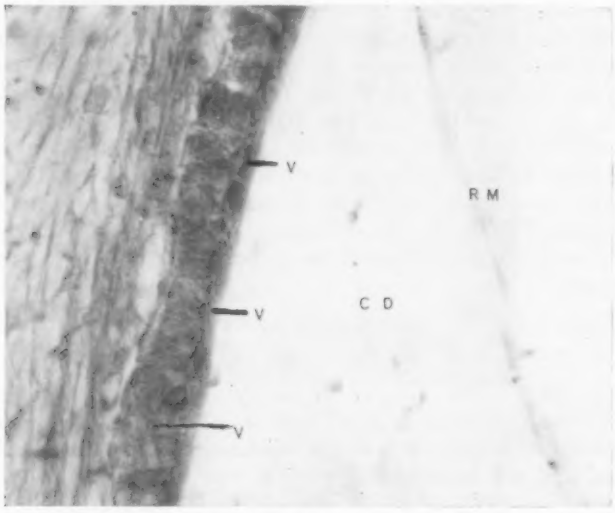

Fig. 4. High power view of cochlear duct. RM—Reissner's membrane; V—strial vessel engorged with blood; CD—cochlear duct containing precipitate with a few blood cells.

SUMMARY OF HISTOLOGICAL FINDINGS.

Fibrinous precipitate was found in the endolymphatic space of each labyrinth except one. In nine labyrinths precipitate was found only in the cochlear duct.

Reissner's membrane was found to be displaced alone, or associated with similar displacement of the basilar membrane in the direction of the scala tympani in every labyrinth. This displacement could not be demonstrated in case the bony labyrinthine capsule was broken during histological preparation.

Engorgement of the large vessel leading to the stria vascularis was clearly visible in six labyrinths.

No blood was discovered in five labyrinths. Few blood cells numbering less than 200 were found near or inside of the round window membrane in ten labyrinths. Rather profuse hemorrhage was seen in the scala tympani of one labyrinth.

CHEMICAL ANALYSIS.

The total amount of perilymph from each ear ranged from 5 to 25 mg. One to 1.5 ml. cc. of cerebrospinal fluid was obtained from each animal. In addition, a sample of blood was taken in each case for correction of blood contamination of the perilymph.

Ultraviolet absorption studies of the perilymph showed a peak at 410, a shoulder at 275, and a sharp end absorption beginning at 265 millimicrons. The peak at 410 should be considered due to hemoglobin contamination. The hemoglobin absorption of the blood sample taken in each individual animal was compared with the hemoglobin absorption of the perilymph, and thus a quantitative measure of the amount of blood contamination was obtained. The shoulder at 275 was considered to be due to tryptophane-tyrosine content of the protein present. Purines and ascorbic acid, however, absorb at this wave length, and were probably present in the fluid (see Table I).

TABLE I—ULTRAVIOLET ABSORPTION OF PERILYMPH IN CATS.

Cat	Conc. %	D(275)	D(410)	Blood D410	D275 Corrected E (1% 1 cm.)
*814	Rt. 16.3	.142	.013*	.49	.008
	Lt. 9.9	.132	.030		.012
815	Rt. 8.9	.327	.212	.38	.028
	Lt. —	—	—	—	—
819	Rt. 13.1	1.04	1.68	.40	.028
	Lt. 5.0	.182	.155		.024
*820	Rt. 8.8	.133	.017*	.41	.014
	Lt. 19.6	.448	.458		.013
*821	Rt. 20.5	.200	.020*	.37	.010
	Lt. 11.7	.197	.078*		.014
822	Rt. 7.6	.450	.578	.44	.026
	Lt. 5.6	.580	.800		.041
823	Rt. 12.2	.630	1.03	.40	.018
	Lt. 11.5	.518	.778		.018
*824	Rt. 4.8	.142	.026*		.027
	1-Lt. 8.1	.510	.660	.45	.026
	2-Lt. 5.8	.345	.500		.020

In specimens marked * there was no microscopic evidence of blood in the labyrinth.

The spectrum of the cerebrospinal fluid revealed only a slight shoulder in the rise of the curve to the end absorption

at 265 (see Table II). It is evident from the data that there are marked quantitative differences in the absorbing substances present in the perilymph and cerebrospinal fluid.

TABLE II—ULTRAVIOLET ABSORPTION OF SPINAL FLUID.

Cat	Conc. %	D(275)	D(410)	D275 / D410 Blood	Corrected E (1% 1 cm.)
814	100	.458	.097	.49	.004
815	8.8	.086	.021	.38	.009
819	100	.318	.023	.40	.003
820	12	.063	.012	.41	.005
821	100	.376	.022	.37	.004
822	10	.147	.047	.44	.012
823	100	.533	.122	.40	.005
824	6.6	.090	.010	.45	.013

In Tables I and II the ultraviolet absorption is given, together with the extinction coefficients E (1 per cent 1 cm.) Correction was made for the blood contamination.

TABLE III—TOTAL NITROGEN IN PERILYMPH OF CATS.

Cat	Weight (mg.)	Total Nitrogen Microgram	Blood Correction (Mg. %)	Corrected N (Mg. %)
814	Rt. 17	11	0	67
	Lt. 16	13	1	73
815	Rt. 7.7	3.9	6	45
819	Rt. 13	14.0	21	85
	Lt. 5.0	2.4	5	42
820	Rt. 8.8	2.9	0	33
	Lt. 21	5.9	4	24
821	Rt. 23	6.5	0	28
	Lt. 12	5.0	0	41
823	Rt. 14	6.4	15	30
	Lt. 13	5.2	12	29
824	Rt. 7.2	5.4	1	73
	Lt. 8.2	7.1	19	68
822	Rt. 7.6	9.6	16	110
	Lt. 5.8	5.1	29	68

After completion of ultraviolet measurements perilymph and cerebrospinal fluid were digested with Kjeldahl mixture. The ammonia in the digest was measured by the microdiffusion method of Conway. Correction was made for blood N in the fluids (see Tables III and IV).

TABLE IV—TOTAL NITROGEN IN CEREBROSPINAL FLUID.

Cat	Weight (mg.)	Total Nitrogen Microgram	Blood Correction (Mg. %)	Corrected N (Mg. %)
814	140	21	0	15
815	88	14	1	15
819	98	11	0	11
820	117	4.8	4	4
821	125	21	0	17
822	17	5.8	1	33
823	121	19	0	15
824	66	5.5	0	8

DISCUSSION.

The presence of fibrin-like precipitate in the endolymphatic spaces in all specimens except one is interpreted as sudden increase in production of endolymph. Marked engorgement of the vessels which lead to the stria vascularis and occasional blood cells in this precipitate (Cat 824 Rt.) point to close relationship between secretory activity and vascular supply (see Fig. 4). There was no evidence of similar precipitate in the perilymphatic spaces (see Figs. 2, 3) which, especially the scala tympani, were primarily involved in the experimental perforation of the round window membrane. Presence of the same precipitate in the saccule (see Fig. 1), utricle and ampullae of the semicircular canals is even more certainly explained by secretory activity, because they are located quite far from the round window membrane, the place of the only injury sustained by the labyrinth *in vivo*. Finally no precipitate was formed in the semicircular canals, the only part of the endolymphatic spaces which does not contain any histological structure capable of secretory activity.

Although no direct evidence has ever been obtained on the location of the source of endolymph, the stria vascularis has been generally accepted as the site of such function (Shambaugh, etc.[8]). The above described histological find-

ings in these experiments are strongly suggestive that similar secretory activity should be ascribed to the maculae of the utricle and saccule, and the cristae of the semicircular canals. Saxen[9] came to a similar conclusion on studying the histology of congenital deafness. Histological findings of Nachlas and Lurie[10] on the stria vascularis of Dalmatian dogs also pointed to the region of the maculae, cristae and the bulb of the endolymphatic duct as possible source of endolymph.

The greatest secretory activity was in evidence in the cochlear duct of the first turn of the cochlea, which is nearest to the round window membrane. The increased secretory flow is interpreted as an effort to equalize the sudden loss of perilymph, which was caused by aspiration through the round window membrane. Replacement of perilymph through the membranous walls of the cochlear duct is possible. Permeability of Reissner's membrane for cristalloids was proven by Gisselsson[11] and Altmann and Waltner.[12] Histological evidence of these experiments, therefore, suggests that perilymph may be replaced by fluid produced in the endolymphatic spaces.

No data were found in these studies which gave any support to the theory of free intercommunication of fluids between cerebrospinal fluid and perilymph. In a few instances blood penetrated from the subarachnoidal spaces into the cochlear aqueduct without reaching the scala tympani of the cochlea. Were the cerebrospinal fluid the major source of perilymph, blood would have been carried into the cochlea by the flow of cerebrospinal fluid.

The minute amounts of perilymph obtainable, and the unavoidable contamination of some of the samples with microscopic amounts of blood, make caution necessary in drawing conclusions based only upon the chemical analysis of the perilymph. Microscopic studies of the ear, in serial sections, in addition to two different analyses of the fluids, ultraviolet absorption and total N, improve the reliability of the conclusions drawn.

It would seem that the amount of blood contamination could be greatly reduced by aspirating perilymph in the dead ani-

mal after blood circulation has stopped, but such procedure would introduce numerous unknown factors; therefore, no reliable conclusions could be drawn as to the normal physiology of the perilymph.

Studies in serial sections failed to reveal any blood in five labyrinths (814 Rt., 820 Rt., 821 Rt., and Lt., and 824 Rt.). These findings fit perfectly into the data of the chemical analysis, and confirm the validity of measurements in other samples slightly contaminated with microscopic amounts of blood. Parallelism between miscroscopic findings and chemical analysis existed to a great degree. A good example is Cat 820; a few blood cells were found under the microscope in the left labyrinth, while there was no evidence of blood in the right labyrinth, thus confirming the ultraviolet measurement. The greatest difficulty in the chemical analysis, contamination of very small samples of fluid by blood, was thus reduced to minimal importance.

Studies in serial sections proved to be very helpful and indispensable to rule out accidental mixing of perilymph with endolymph through an artificial tear of the membranous labyrinth.

The great difference between the protein contents of the perilymph and cerebrospinal fluid disproves previous concepts of identity of these two fluids. In the light of the same data, a free flow of cerebrospinal fluid through the cochlear aqueduct into the scala tympani is equally untenable.

No significant difference in the protein contents of perilymph was found between samples taken before or after removing cerebrospinal fluid.

In a few instances the sodium content was measured with a flame photometer, and was found to be almost identical in concentration in the perilymph and cerebrospinal fluid. Sodium was selected for measurements because it stays mostly in the interstitial tissue fluids; thus it is most likely to take part in diffusion or ultrafiltration.

The greater concentration of protein found in the perilymph of humans suffering from Meniere's disease, as com-

pared with the cerebrospinal fluid of the same patients, is most probably not pathological in nature. Similar quantitative differences exist between the cerebrospinal fluid and perilymph of normal cats.

Further study of the nature of human labyrinthine fluids would offer valuable information. Ectasia of the endolymphatic spaces in Meniere's disease certainly points to a disorder of the fluid system. A sizable group of unexplained cases of deafness, especially those with sudden onset, may well benefit from better knowledge of the labyrinthine fluids.

SUMMARY.

1. Measurements of the ultraviolet absorption and total nitrogen were made on the perilymph and cerebrospinal fluid of 30 cats.

2. The total N concentration was more than twice that of the cerebrospinal fluid taken from the same animal in every instance.

3. Microscopic studies of sixteen labyrinths were done in serial sections. There was no evidence of microscopic blood contamination in five labyrinths, while only a few blood cells were present in ten.

4. Blood correction in the chemical analysis proved to be accurate when data were compared with microscopic findings.

5. Mixing of the perilymph with endolymph was ruled out by microscopic studies.

6. The great difference in chemical composition between perilymph and cerebrospinal fluid rules out all possibility of free flow of fluid between subarachnoidal and perilymphatic spaces.

7. The greater concentration of proteins in the perilymph of humans suffering from Meniere's disease is most probably not pathological in nature, in view of the above findings.

8. Histological studies of the same animals point to the increased secretory activity in the endolymphatic spaces following drainage of the perilymph through the round window

membrane. This again is interpreted as indirect evidence of the role of the endolymph as source of perilymph.

9. Localized precipitation of endolymph in the cochlear duct, in the saccule, utricle and ampullae of the semicircular canals is considered as indirect evidence of increased production of endolymph in these anatomical structures.

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THE INARTICULATE CHILD: A PROBLEM IN DIAGNOSIS.*

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Although the title of this paper might suggest that the approach to the inarticulate child is academic and no more, that inference would be entirely incorrect. The state of these children is always distressing, and so is the state of their parents. The approach to the problem, for a problem it is, while it must be compassionate, must also be scientific. Whether these children, in spite of their handicaps, are to lead happy, successful and useful lives depends, first of all, upon a precise determination of the nature of their handicaps. An inarticulate child is not necessarily deaf, nor is he necessarily dumb, in the dictionary sense of being unable to speak or in the more colloquial sense of the term. He may be aphasic because of brain damage or for other reasons; he may not talk, or his speech may be incomprehensible for psychological or functional reasons.

How the inarticulate child shall be treated depends upon the identification of the special cause of his inarticulateness, What his future life is to be depends upon how he is treated. An enormous responsibility, therefore, rests upon the otologist when once the parents bring the child to him. I might go further and say that an equally enormous responsibility rests upon the general practitioners and the pediatricians, who so often see these children first, and by whose advice the parents are guided in what they do next, and how soon they do it.

The inarticulate child is more frequently encountered today than he ever was before, for two reasons: first, that more newborn children are surviving the difficulties of birth and of early prenatal life. Rh incompatibility in the parents is approaching control. Maternal toxemia and the hemorrhagic

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complications of pregnancy are also approaching control. The child himself, thanks to improvements in therapy, including modern antibiotic therapy, is surviving such illnesses as encephalitis and meningitis, which formerly had a very high mortality and which still leave residua, among which deafness is prominent.

The second reason why we are seeing more inarticulate children today is that they are being referred to us with much greater frequency. Some errors are still being made, but I am glad to say that the day has almost passed when pediatricians, general practitioners, and even otolaryngologists would tell the parents that a child is too normal otherwise to have anything wrong with his hearing, or that he will probably outgrow his unawareness of sound, or that, if his hearing is really impaired, he must be five or six years old before auditory testing is possible. It is never too early to start testing and training the deaf or otherwise inarticulate child. It is also never too late, but the end results are less good than when the start is made early. For one thing, the child whose impairment is slight or even moderate can frequently be so rehabilitated as to keep up with normal children. Schools for the deaf serve an invaluable purpose, but a normal life with normal children is better. For another thing, as Ewing and Ewing[1] emphasize, interest in persons is an essential foundation of the acquisition of lip reading and speech training. It is, therefore, far better that such training should begin while the child is still in close physical contact with his mother and his nurse, and has not yet become deeply concerned with physical things.

In recent years there has been a great deal of active emphasis on testing of the hearing of infants and young children. This is as it should be. Our methods, however, still leave something to be desired. One might gather, from the medical literature and from presentations at medical meetings, that the only techniques which provide satisfactory diagnostic information are such complicated ones as psychogalvanic skin-resistance audiometry (PGSH), electroencephalography, or pure tone audiometry by peep-show methods. This is far from the truth.

These are excellent tests, but only in their places; they are not infallible, they are merely adjuncts to more basic procedures. The equipment is expensive, and special training is required to operate it. These are valuable methods in speech and hearing centers, but they are not suitable for use in the average otolaryngologic practice, and they are, of course, completely impractical for use in the office of the general practitioner who, either because of desire or necessity, combines otolaryngology with general practice. Finally, they are not methods in any way suitable for use in testing very small children.

One of the most unfortunate results of the impression that these diagnostic methods are necessary for diagnosis, is that so many physicians apparently believe it necessary to refer the inarticulate children to speech and hearing centers in which they are available. Lest I be misunderstood, I hasten to say that I have much to do with the operation of the speech and hearing center at the Tulane University School of Medicine, where equipment for these tests is available, but where they are seldom carried out on very young children.

A second unfortunate result of the impression that these tests are essential for diagnosis in children is the adoption of this point of view by residents in otolaryngology. It is the same sort of error, of course, which leads residents in other specialties, who all too often have the bad example of their elders to guide them, to substitute laboratory tests for the use of simpler clinical methods, often to the detriment of their patients. Residents of otolaryngology, like the other residents, must be brought back to first principles. In the diagnosis of hearing and speech difficulties, these principles are, in this order, history, clinical observation, and clinical evaluation, with testing last of all.

DIAGNOSTIC CONSIDERATIONS.

For a long time it was assumed that hearing tests which had been found satisfactory for adults were equally satisfactory for children. They are not. As Myklebust[2] has well expressed it, the evaluation of hearing in adults who are emotionally, intellectually, physically and perceptually mature, is a very different process from its evaluation in children who are none of these things. An experience recorded by Ewing

and Ewing[1] makes this clear. They found that three children who were apparently totally deaf when tested with loud sounds, responded to instructions spoken in a moderately loud voice at a distance of six feet.. Obviously, factors other than the purely physical ones of pitch, loudness and duration of sound may determine the young child's response or failure to respond to it. Obviously, the whole basis of tests of hearing in young children requires re-evaluation.

Pure tone audiometry has proven an extremely useful method for diagnosing hearing losses in adults. In children, as most of us realize, pure tones are not suitable auditory stimuli. The child does not readily respond to them, because they are beyond his perception. He is much more likely to respond promptly to mixed sounds at the same level of intensity, or even at lower levels. My own experience agrees with that of other observers: reliable responses to pure tones are unlikely to be achieved until the general mental and perceptual level is that of the average normal five to six-year-old child. The important consideration here is that five or six years of age is far too late to diagnose a hearing loss or speech difficulty and to do something about it.

Modifications of pure tone audiometry are also unsatisfactory in young children. The PGSR test has two objections: first, that it is dependent upon pure tone stimuli, and these, as has just been pointed out, are not reliable at this age; second, the technique has not been standardized on young children with normal hearing, and there are, therefore, no criteria to evaluate hearing losses in abnormal children. Still another objection might be mentioned, that if the child is emotionally disturbed, any test that depends upon a conditioned reflex to a disagreeable stimulant may indicate a hearing loss when none is present, or a greater hearing loss than really exists.

When the peep-show technique of pure tone audiometry[3] is used in children over five years of age, the results are satisfactory, and a threshold test can be quickly run. Between the ages of three and five years, however, the technique, ingenious as it is, is not entirely reliable, and the results are not commensurate to the time spent on it. Below the age of three years it has been completely useless in our hands.

As to the electroencephalograph, it is of undeniable value in revealing brain damage or in excluding it. Special techniques have been developed for use in children when they are asleep,[4] but we prefer to use it with noise makers in a free field. It often helps to explain delayed, retarded or unintelligible speech in a child with no hearing loss or only slight loss. It remains, however, an adjunct method, which does not supplant basic clinical principles.

Diagnosis in the preschool child is time-consuming and frustrating. More often than not, as Bordley[5] says, it results in the otologist's being more hopeful that he is right, than being sure that he is. The more carefully the investigation is conducted, however, the more likely he is to be right.

Before he attempts to diagnose hearing or speech difficulties in young children, the otologist must have clearly in his mind the stages of normal development:

The normal child sits alone at five to seven months and crawls between eight and ten months, unless he skips the crawling stage and begins to walk at once. Ordinarily, however, he walks between 12 and 18 months. Toilet training for the bowel occur between 15 and 24 months, and for the bladder between 18 and 24 months during the day, and between 24 and 30 months at night if he is taken up.

If hearing develops normally, it will be according to the following schedule:[6]

There is some response to sound immediately after birth, which becomes more definite at one month. The response takes the form of a reflex, which is usually palpebral. During the first three months of life infants respond more readily to noise makers than to the voice.

During the fourth through the sixth months the voice is at least as good as a noise maker as a form of auditory stimulation. A quiet voice is more effective than a loud voice.

During the second half of the first year of life the child continues to respond to noise by reflex reactions. He becomes adept at recognizing meaningful sounds, such as tapping on the nursing bottle. He begins to localize sound by turning his

head or his eyes to the source. Certain voices quickly attract his attention.

During the second year of life the child responds to simple speech tests. The whispered voice is unsuitable for testing at this age, and a quiet voice continues to attract his attention more quickly than a loud voice.

During the third year of life the child shows a great capacity to localize sound. Speech and voice tests are now a satisfactory method of testing hearing.

Between the ages of three and five years the normal child responds to vocalizations and whispers; he will also respond to pure tone audiometric testing, though as a rule at least another year must pass before this technique gives entirely satisfactory results.

The normal child, if a girl, will usually begin to say single words between 10 and 12 months. A boy does not usually begin to talk until several months later, when he is 14 to 16 months. Both use two-word sentences between 18 and 24 months.

It is now known that during the first 12 to 18 months of life there is no difference in the vocalization of the normal child and the deafened child, even when the deafness is severe. The deaf child's voice has natural quality and inflections, and skilled training, if begun early, can do much to conserve the natural properties.

It is extremely important that the otologist should thoroughly understand these normal stages in the child's development. It is equally important that he realize that the times just stated vary widely in individuals. On the other hand, the child who has not said a single word up to the time he is 16 months old, or who has showed no response to sound by the time he is 12 months old, needs medical consultation, and needs it promptly. The first three or four years of life are the critical ones in the development of any child, and they are even more important in the child whose speech and hearing are impaired to any degree at all.

ROUTINE OF INVESTIGATION.

To turn now to the positive side of the problem, the investigation of the inarticulate child begins with a complete pre-

natal and postnatal history, careful observation, evaluation of the findings, and then, last of all, actual testing of the hearing. All of this takes time, and nothing is gained by trying to make haste or to use short cuts; furthermore, the assistance of the mother is imperative in the investigation, and she will withdraw into herself if the physician is not considerate, thoughtful and sympathetic. Incidentally, when the investigation is concluded, he must take the time to explain the situation to her in the simplest possible manner, and to give to her as much hope as the child's condition warrants.

History Taking: Our own procedure, when an appointment is made for an inarticulate child, is to defer it for several days, in order to give the parents time to fill out the questionnaire, which is immediately mailed to them, and to return it to us for review and analysis before the appointment. It is sometimes difficult to secure an exact statement from the mother as to the child's difficulties when the question is abruptly broached to her at the office, nor is the other information she supplies always prompt and accurate. Given the time, however, she will clarify her own thoughts by writing down the replies to the questions asked in the history sent to her, and the data will be as full as can possibly be secured.

If no appointment has been made, a short history form is used in the office at the first visit, but it is not so satisfactory.

The questionnaire mailed to the mother provides for detailed information concerning the family history; the prenatal history; the delivery; the diseases, operations and accidents of postnatal life; and the story of the child's development and general progress, as well as his progress in speech and hearing. Specifically, the following points are covered, in addition to details of the family history and maternal history: When the speech or hearing difficulty was first noticed. Precisely what form it takes. Whether other difficulties are also present, such as visual impairment or behaviorism. The weight and respiration at birth. Feeding problems. All the possible illnesses of childhood and their complications. When the child first sat, walked, fed himself, was toilet-trained, dressed himself, said words and made sentences. Handedness. Appetite.

Habits of sleep, including bed-wetting. Other habits, such as sucking the thumb and biting the nails. Temper tantrums and destructiveness.

Questions are asked as to how the child compares with other children in progress and behavior, whether he can convey his meaning, whether he can understand his mother, whether he prefers the father or the mother, and how he reacts to people in general. In short, there is almost no conceivable piece of information which this form does not provide for, and parents are usually most cooperative and helpful in filling it out.

Clinical Observation: When the child is brought to the office, all the circumstances are kept as normal as possible. The young child may have recollections of other visits to a physician's office and of unpleasantness associated with them, and such memories would hinder the investigation if they were brought back to him. The physician, therefore, removes his head mirror and replaces his white office coat with his ordinary coat. After a casual greeting, the child is allowed to move about as he pleases while the physician discusses with the mother the data she has supplied, or while a brief office history is taken if the longer form has not been filled out. During this time the child is unobtrusively observed.

The hearing of young children can be tested in various ways. Our own preference is for the use of noise makers in a free field. The equipment which we use, and which is purchased from a music supply house, makes sounds between the 200 and 5,000 frequencies. The noise makers are checked as to frequency or frequency range with an octave analyzer on a sound level meter, after which it is possible to produce sounds of the decibel or power level desired and to record the results accurately. We begin with a tone around the 25 to 30 decibel level, since it has been established that many children will respond to a tone at this level and not to one at a higher level. If no response is elicited, the sound is increased until it is heard or it is clear that no sounds can be heard.

As has been pointed out, we try to make the conditions of the test as natural as possible. For all practical purposes,

testing is by the so-called cessation of activity technique, which, as the name implies, means that the child ceases whatever he has been doing because a sudden noise is impressed upon his consciousness. The windows in our office overlook the Mississippi River, and there is a great deal of industrial and shipping activity on it at all times. Testing can often be carried out while the child is absorbed with the many sights along the river. If he is not interested in these sights, or is too small to be interested in them, he is given some type of toy, most often a ball, to occupy him. The very young child is permitted to have his bottle.

Eventually a detailed investigation of the nose, nasopharynx and Eustachian orifices must be carried out with the electropharyngoscope, but this is always deferred until all other clinical observations have been made. If it were the first phase of the investigation it would obviously end all hope of rapport with the child. Laboratory tests of hearing are also deferred until clinical observations are concluded.

DIFFERENTIAL DIAGNOSES.

The differential diagnosis of inarticulateness is usually possible by these simple clinical methods if enough time—it takes a great deal—is devoted to them, and if the observation is careful and thorough. All of the facts which are stated hereafter, can be secured in this manner.[6] When they are properly evaluated, the cause of the child's failure to speak, or of his poor speech, is usually evident.

Response to Sound: The child with impaired hearing responds to sound consistently at the threshold level and age level. If he can hear a sound at 30 db or less, or if he can hear conversational speech, his inarticulateness cannot be attributed to inability to hear.

The child who does not respond to sound until it falls between 30 to 60 db is suffering from a moderate hearing impairment, frequently of conductive origin, and usually correctible. Until the correction is made, however, he cannot acquire normal speech because he does not hear normal conversational tones. If the loss is no more than 40 db, and he is taught lip reading, he can remain in regular schools provided with special seating arrangements and similarly aided.

If the child responds to sounds from 60 to 100 db, his hearing loss is probably on a perceptive basis, and speech can be heard only if it is amplified. If there is no response to sounds of less than 100 db intensity, the hearing loss is extremely serious. Hearing aids will be of no value and schooling in special schools for the deaf will be required.

In contrast to the child with impaired hearing, the aphasic child does not respond to sound consistently. The emotionally disturbed child is apparently willful in his failure to respond. The mentally deficient child responds consistently to the extent permitted by his mental capacity; his response is usually to sounds which indicate food and similar things to him.

Visual Clues: The child with impaired hearing relies heavily on vision and is, therefore, unduly sensitive to hearing and other visual clues. The aphasic child, because of central nervous system damage, either does not rely on vision at all or does so erratically and intermittently. The emotionally disturbed child does not rely on vision, since his disturbance is generalized in effect. The mentally deficient child need not rely on vision, for he is using his auditory sense to his full capacity, and, therefore, has no loss for which to compensate.

Speech: The child with impaired hearing acquires a speech pattern consistent with his capacity to hear. He does not vocalize for pleasure but uses sound with intent and purpose, in a logical manner, to express his feelings and needs. Echolalia (parrot or echo speech) is not present. The impairment of the quality of the voice depends upon the hearing loss.

The aphasic child attempts to speak, but the result is jargon, which is spontaneous but meaningless. He uses his voice playfully or indiscriminately, without meaningful intent or purpose. Echolalia may be mild, moderate or marked. The tonal quality of the voice remains about normal.

The emotionally disturbed child seldom speaks, and is frequently completely mute. He does not usually use his voice for pleasure or to attract attention, though in less severe cases the voice is sometimes used in play, but always erratically. He does not use jargon. Echolalia is present in severe emotional deviations. The tonal quality is normal.

The mentally deficient child acquires speech eventually but at a later age than normal. He uses his voice for pleasure and projectively at the level of his mental capacity. Echolalia is not present. The tonal quality does not deviate from normal.

Use of Gestures: By the age of two years the child with impaired hearing uses gestures himself and understands those of others. The aphasic child does not use gestures effectively. Sometimes he is unable to make them. In other instances he uses them frequently but inconsistently. The emotionally disturbed child, who has rejected his environment, does not use gestures at all. The mentally deficient child uses them in accordance with the stage of his mental development.

Laughing, Smiling, Crying: The child with impaired hearing smiles and laughs less than the normal child. He cries often, and with vigor and feeling. The aphasic child does not laugh or smile freely, though he may laugh compulsively, in a defensive manner. His cry is typically a whine. The emotionally disturbed child does not laugh or smile except in a preoccupied or disturbed manner. He does not share the laughter of others. He cries without obvious reason. The mentally deficient child laughs, smiles and cries normally so far as tone is concerned, but his reactions are delayed.

Other Senses: The child with impaired hearing has no disturbance of visual perception, and the same is true of the emotionally disturbed child. The child with aphasia frequently misperceives both objects and sounds. The mentally deficient child frequently misperceives sounds but not objects, his perception being, on the whole, commensurate with his mental capacity.

The child with impaired hearing is usually sensitive to tactile sensation, relying on his sense when the visual sense is in use. The aphasic child is not unduly responsive to tactile stimuli and tends to ignore them while he is engaged in activity. The emotionally disturbed child is generally unresponsive to such stimuli. He acts as though tactile sensations were nonexistent. The mentally deficient child is not unduly responsive to them, since he is using all his senses to the best of his ability.

Motor Behavior: The child with impaired hearing sits and walks normally at the usual age. He is hyperactive, but his activity is consistent and meaningful. His balance may be poor and he tends to shuffle as he walks. The emotionally disturbed child may be late in sitting and walking because he does not want to sit or walk. He may show stereotyped activities, such as rocking or twirling. The mentally deficient child is slower in sitting and walking than children in the other groups and is retarded in all other motor activities.

Attention to Facial Expression of Others: The child with impaired hearing is unduly responsive to the facial expressions of his associates. The aphasic child is incapable of the sustained attention necessary to profit from them. The emotionally disturbed child, as a manifestation of his rejection of the world, ignores face to face conduct. The mentally deficient child is realistic to the extent of his capacity to profit from attention to the facial expressions of others.

Emotional Expression and Development: The child with impaired hearing is immature and delayed emotionally, but is otherwise normal in this respect. The aphasic child is immature emotionally. Such emotions as he displays are superficial and he has no basic means of expressing them. The emotionally disturbed child does not show normal response to affection. He does not laugh, cries very little, and apparently has no ability to express emotion. The mentally deficient child is more immature emotionally than the child with impaired hearing. His emotional expression is in proportion to his mental age.

Social Perception: The child with impaired hearing is somewhat less efficient in social perception than the normal child. He relies entirely on vision. He is normally shy and reticent. The aphasic child, who is incapable of understanding social situations, attempts to relate them to his environment. He has no shyness or reserve. The emotionally disturbed child behaves as he pleases. He regards people as objects and does not include them in his world. The mentally deficient child is aware of people around him and accepts them at the level of his mental capacity.

Perseveration, Distractibility, Disinhibition: These qualities are not observed in the child with impaired hearing. They exceed normal expectation in the emotionally disturbed and the mentally deficient child, and are very marked in the aphasic child.

SUMMARY.

The inarticulate child is being referred to the otologist more frequently than ever before, but the belief persists in many quarters that he must be investigated by elaborate laboratory methods. Actually, pure tone audiometry, PGSR audiometry, audiometry by the peep-show technique, and electroencephalography are not suitable for diagnosis of speech and hearing defects in young children. Instead, diagnosis is accomplished, and in this order, by detailed history-taking, clinical observation, and clinical evaluation, with laboratory investigation last of all. The use of noise makers in a free field, by the so-called cessation of activity technique, is a desirable method of testing hearing in young children because it can be done unobtrusively, in natural circumstances.

A technique of history-taking, auditory testing and clinical evaluation of the total child is presented, and the criteria of differential diagnosis are briefly outlined.

The approach to the problem, while it is entirely scientific, does not forget that both the child and his parents are human beings in distress, who need all of the physician's understanding and compassion in addition to his technical skill.

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251 Third Street.

SYMPOSIUM:

BRONCHOGENIC CARCINOMA.

I—SUSPICION.*†

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In preliminary summary, certain general facts pertinent to the subject of bronchogenic carcinoma are deserving of comment. There is evidence that the actual incidence of the disease appears to be increasing, particularly in men. Recent advances in thoracic surgery, yielding low mortality rates, render exploration of the chest and surgical treatment of the disease by excision both safe and effective. The ability to cure patients suffering from bronchogenic carcinoma, however, depends upon prompt surgical treatment; thus, early recognition based upon suspicion yields the most productive results in the control of bronchogenic carcinoma. Herein lies the great opportunity for the physician who first sees the patient, whether he be otolaryngologist, internist, or general practitioner.

Unfortunately, there are a number of factors which interfere with successful treatment of bronchogenic carcinoma. These could be classified as the factor of the disease itself, the factor of the patient, and the factor of the physician. The factor of the disease itself can be illustrated in the following fashion. While it is a recognized fact that early bronchogenic carcinomas are generally discovered in routine thoracic roentgenograms and that their successful treatment has followed such discovery, even under serial roentgenographic observation hopeless situations have arisen. The following report of a case is cited:

Case 1: A 63-year-old furniture factory worker and former miner had been examined over a period of two years by the chest clinic of a city

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(a) (b) (c)

Fig. 1: a—Chest film of a 63-year-old man showing bilateral apical lesions considered to be consistent with the diagnosis of silicosis. This film represents the last of four films taken over a period of two years, all of which demonstrated stability of the apical lesions. b—Subsequent film of same patient revealing a large left apical density. This film was made less than one year after that shown in a. c—Anteroposterior view of cervicothoracic spine of same individual. A destructive lesion of the left second thoracic transverse process is shown.

Public Health Service. Typical nodular lesions of silicosis were found in the X-ray film of the chest (see Fig 1a), and repeated X-rays eventually revealed stability of these lesions. The last film was taken three months prior to the onset of the patient's illness, which was ushered in by pain in the left forearm and hand, and later by the development of a left Horner's syndrome. A roentgenogram taken several months following the onset of the illness revealed a left apical intrathoracic tumor, (see Fig. 1b); and in a film of the spine destructive lesions of the transverse processes and bodies of the first and second thoracic vertebrae were seen (see Fig. 1c). In an attempt to arrive at a diagnosis, a supraclavicular and an axillary lymph node were removed. Neither of these nodes manifested abnormalities which would suggest the nature of the left apical tumor. Successful biopsy, however, was performed by means of a needle inserted into the region of the transverse process of the first thoracic vertebra. Microscopic examination of the tissue removed revealed a well-differentiated squamous cell carcinoma. The findings in this case were consistent with the diagnosis of a superior sulcus tumor that was inoperable because of neural and osseous involvement.

It is highly unlikely that any available methods of the present day could have led to a satisfactory outcome in the case of this particular patient. One cannot say that unavoidable delay existed in the diagnosis.

The factor of the patient in terms of an interfering factor in the successful treatment of bronchogenic carcinoma can be expressed as follows: Either the patient fails to seek medical aid in the presence of evident symptoms, or he refuses to follow worthy advice. Convincing evidence comes from a series of ten patients reported on by Abeles and Ehrlich.[1] All of these patients had solitary undiagnosed pulmonary lesions discovered in routine thoracic roentgenograms. All had no symptoms and all refused exploratory thoracotomy. Within a year of observation five of the group exhibited evidence of spreading intrathoracic malignancy.

The third interfering factor is that of the physician himself. Countless examples can be found to illustrate that physicians unintentionally have followed a program of watch and wait rather than leading patients directly to definite diagnostic steps, even to the extent of exploratory thoracotomy. In the course of the past ten years two factors have become widely recognized; namely, that conventionally recorded signs and symptoms of bronchogenic carcinoma are of little value in the recognition of the disease in its early phase, and that pulmonary cancer is the only visceral malignant lesion which casts a shadow in an ordinary roentgenogram. The foregoing two points indicate action consisting of encouraging patients to

have X-rays of the chest made at intervals of at least once a year or even more frequently. Obviously there is a certain practical limit. In the management of adult patients it is not unreasonable to insist upon X-rays at annual intervals and X-rays of the chest whenever respiratory symptoms appear.

There are certain situations in which lie opportunities for early diagnosis of bronchogenic carcinoma. Some of these can be listed as follows: The solitary circumscribed pulmonary lesion, the patient with hemoptysis, the patient with pneumonia, and the patient in whom one discovers a localized wheeze. Because of the frequent occurrence of inflammatory pulmonary lesions resulting from bronchial obstruction due to bronchogenic carcinoma, an illustrative report of a case follows.

Case 2: A 62-year-old white carpenter was first admitted to the hospital with the complaint of cough, hemoptysis and dyspnea for ten days. The patient was febrile and a roentgenogram (see Fig. 2a), disclosed changes consistent with atelectasis in the lower portion of the right upper lobe. On treatment consisting of penicillin intramuscularly the patient's symptoms rapidly disappeared. A roentgenogram taken approximately two weeks later revealed persistence of the lesion in the lower portion of the right upper lobe. Because of failure of this lesion to clear and because of persistence of cough, bronchoscopy was advised.

This procedure revealed no evident bronchial lesion. Cytologic studies of the sputum revealed no evidence of neoplastic cells, and no organisms of tuberculosis were seen in specimens of sputum. Skin tests for coccidioidin and histoplasmin were negative and a tuberculin reaction was positive in the second strength. Approximately one month after the onset of the patient's initial illness, during which time diagnostic studies were carried out, he was found to have a wheeze in the region of the right upper lobe. At this time the roentgenogram had not changed and exploratory thoracotomy was undertaken.

At operation a small bronchogenic carcinoma was found somewhat distal to the origin of the right upper lobe bronchus. This lesion had produced no ulceration, but it had produced a moderate degree of bronchostenosis. There was no evidence for metastatic spread of the lesion to the hilar lymph nodes and the lesion itself was extremely small, measuring less than 2 cm. in diameter. Total right pneumonectomy was performed and all mediastinal nodes were removed. At the time of thoracotomy it appeared that the end-result would be quite favorable in this instance (see Fig. 2b).

Because of the variable behavior of bronchogenic carcinoma, several features of the disease are worthy of comment. The characteristics of behavior which are of great interest

are those which produce subtle changes in the thoracic roentgenogram. Outstanding are abnormalities of the pulmonary hilum. An example seems indicated.

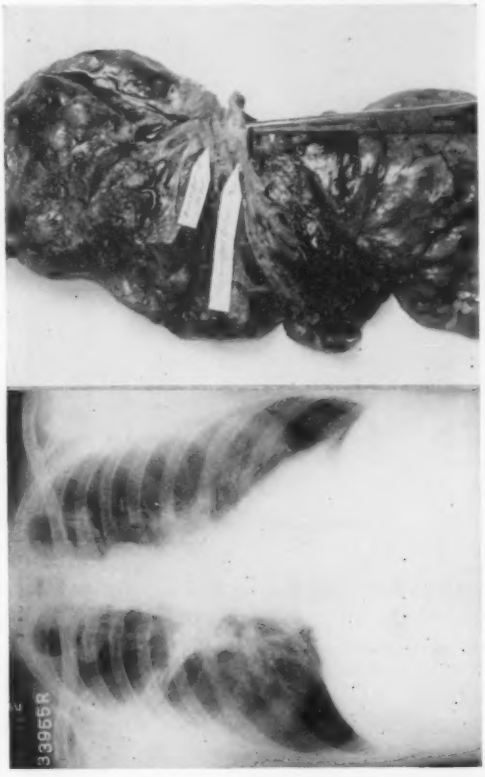


Fig. 2: a—Thoracic roentgenogram of a 63-year-old man with a history of cough, dyspnea, fever, and hemoptysis of 10 days' duration. The lesion in the right upper lobe failed to clear, and one month after onset of symptoms a wheeze was audible over the right upper lobe. b—Small bronchogenic carcinoma in right lung removed at operation. Resected hilar lymph bodies were free of metastases.

Case 3: A 59-year-old patient was treated for arteriosclerotic heart disease with angina pectoris for a year. A roentgenogram of the thorax had been taken initially. This is shown in Fig. 3. If one examines it closely one can detect a slight abnormality of the left hilum between the aorta and the pulmonary artery. During the period of treatment of his heart disease, the patient developed enlargement of a left supraclavicular lymph node. When this node was removed for microscopic examination, it was found to contain squamous cell carcinoma considered to be

metastatic. It was the opinion of the pathologist that the lesion of the left hilum represented the primary tumor arising in the bronchus.

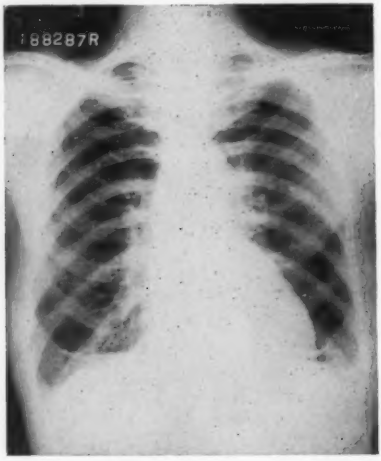


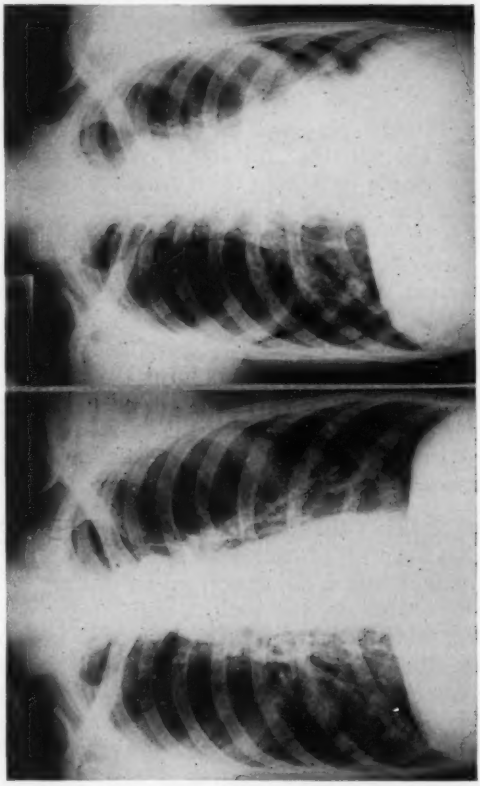
Fig. 3: Thoracic roentgenogram of a 59-year-old man which manifests slight abnormality of the hilar structures in the region between aorta and pulmonary artery. One year after the film shown was taken the patient was discovered to have an enlarged left supraclavicular node. This node was removed for microscopic examination, with the discovery of metastatic squamous cell carcinoma.

This patient exemplifies the fact that very subtle changes in the contour of the hilar shadow should arouse suspicion of bronchogenic carcinoma.

A second subtle change occasionally seen is that of minor shift of the mediastinum, particularly when the cardiac shadow occludes visualization of atelectasis in the basal segments of the left lower lobe.

A third circumstance that should arouse one's suspicion of bronchogenic carcinoma is that of localized emphysema. Fig. 4a shows a film that appears to be within normal limits. It was taken in full inspiration. At *b* in the figure is a film taken in expiration. It demonstrates that the right chest remains inflated during expiration. This of course is diagnostic of

obstruction to the main stem bronchus. In this particular instance a bronchogenic carcinoma was discovered in the bronchus to the right lung.



(a) (b)

Fig. 4: (a)—Thoracic roentgenogram taken in full inspiration. (b)—Thoracic roentgenogram of same patient taken in full expiration. Obstructive emphysema of the right lung is demonstrated. This was discovered to be caused by bronchogenic carcinoma involving the right main stem bronchus.

A fourth situation which should arouse one's suspicion is that of the patient with pleural effusion that eludes diagnosis. Peripheral carcinoma of the lung often presents itself symptomatically by the production of pain of pleural origin accompanied by pleural effusion. The traditional diagnostic thought that tuberculosis is a cause of most unexplained pleural effu-

sions must be abandoned. The mere presence of fluid does not mean hopeless pulmonary cancer.

SUMMARY.

In recent years advances in thoracic surgery have made possible extirpative treatment of bronchogenic carcinoma; hence in the light of present knowledge, successful treatment of the patient with bronchogenic carcinoma depends upon early recognition of the disease. This latter can be accomplished only through suspicion on the part of the physician who first sees the patients. An active role can be played by the physician by his insistence upon frequent thoracic roentgenograms for his patients. Unfortunately, there are certain factors which interfere with successful therapy. These consist of the factor of the disease itself, which can be expressed by the statement that bronchogenic carcinoma can progress to an inoperable stage even though the most careful observations are carried out. A second interfering factor is that of the patient himself. This is exemplified by the patient who refuses to follow advice or who ignores obvious symptoms. A third interfering factor lies with the physician himself who fails to lead the patient the full diagnostic route, including surgical exploration of the chest.

There are certain situations in which lie opportunities for the early diagnosis of bronchogenic carcinoma. These consist of the patient with a circumscribed pulmonary lesion, the patient with hemoptysis, the patient with pneumonia which fails to clear, and the patient with a localized wheeze. Certain behavior characteristics of bronchogenic carcinoma which are of diagnostic interest were mentioned. These consist of abnormalities in the hilar area, slight shift of the mediastinum, localized emphysema, and unexplained pleural effusions.

It is emphasized that the responsibility for the early diagnosis of bronchogenic carcinoma lies with the physician who first sees the patient. Suspicion on his part can lead to successful treatment of bronchogenic carcinoma.

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SYMPOSIUM:
BRONCHOGENIC CARCINOMA.
II—RECOGNITION.*

John F. Tolan, M.D.,
Seattle, Wash.

Benign and malignant tumors produce pulmonary findings characterized for the most part by their location, by the degree of obstruction, and by their sojourn, rather than by their histologic classification. Benign tumors of the bronchial wall, such as polyps, lipomas, papillomas, fibromas and chondromas may give an identical clinical picture to that of a bronchogenic carcinoma; however, in many cases, if there is a minimal degree of complicating bronchopulmonary suppuration, the benign character of the lesion is apparent by the lack of infiltration or destruction of the adjacent bronchial walls.

Bronchial adenomas must be considered as special tumors, exceedingly slow growing, somewhat invasive in character. Their appearance through the bronchoscope is that of a smooth, round mass which is firm, freely bleeding and often covered with a smooth, vascular appearing membrane. Endoscopic removal is sufficient to cure the disease in approximately half of the cases; however, if there is local recurrence, if the tumor bleeds excessively on biopsy, or if there is lung destruction distal to the tumor sufficiently great to necessitate surgical intervention in itsef, surgical removal of the tumor and of the area of lung it has obstructed is indicated.

Closely related to the adenomas, but a distinctly different clinical entity are the cylindromas of the bronchus. These are malignant and, while they, too, are slow growing, they metastasize to the regional lymph nodes and other areas of the body. Therapy here is entirely surgical, although palliative removal of obstructing tissue may be indicated if the position of the lesion indicates it to be nonresectable.

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At present, there are scores of laboratory tests for the determination of the presence of cancer. Few are reliable, and none is sufficiently accurate to suffice without co-ordination with other diagnostic procedures. The most satisfactory results in the cytological diagnosis of pulmonary cancer have been obtained by the examination of material aspirated from bronchi during the course of bronchoscopy. Although collection of bronchial secretions requires bronchoscopy, this is not a real disadvantage inasmuch as all suspected cases of lung cancer should be bronchoscoped.

The location of the site of origin determines whether or not an early lesion may be seen and biopsied bronchoscopically. In advanced cases, the growth may propagate along the bronchial wall and bring it nearer the hilar area. In the great majority of cases in the late stages, the tumor will be visible, and most are accessible for biopsy. In early cases, however, only those that originate in the lobar or first portions of the segmental bronchi will be visible. Telescopic-lens accessories that permit better visualization of segmental bronchi in all lobes are available.

Bronchoscopy serves another purpose: in addition to determining the identity and location if a lesion is visible, it permits evaluation of the status of the airways to the involved lung. If excisional therapy is in the plan, it is essential that everything possible be known about that part of the bronchial system that supplies the sound lung. For diagnosis, bronchoscopy serves in three ways: visualization of the abnormality, provision by biopsy of material for microscopic study, and aspiration of secretions for cytological examination. It must be remembered that in cases of early cancer the majority of examinations will be visually negative.

Bronchogenic carcinoma assumes many variable characteristics. The most common endobronchial form is the soft verrucose, bulky tumor obstructing a major bronchus, or the bronchus to a lobe or a segment. It is red, friable, freely bleeding, and behind it lies atelectatic lung and broncho-pulmonary suppuration in the form of bronchiectasis or lung abscess. The tumor may be covered with exudate, necrotic debris or purulent secretion, which must be aspirated carefully before tissue is

taken. Other tumors are polypoid in character with a mulberry-like appearance. Some carcinomas invade the bronchial walls submucosally, or through the peribronchial lymphatics, to produce an extreme induration of the bronchial walls far above the point of the complete bronchial obstruction, which was determined as the location of the lesion by physical and X-ray findings. Occasionally an ulcerative type of lesion is found with rounded edges and a necrotic base extending along the floor or wall of the bronchus. Such lesions are not often seen bronchoscopically, because at this stage they produce relatively few symptoms which call attention to their presence.

Bronchogenic carcinoma also may be found as a small thickening of the bronchial wall with no bronchial obstruction and no pulmonary findings physically or by chest X-rays. Such tumors metastasize extensively and death resulte from the widespread metastases rather than from the primary tumor. Only the postmortem examination reveals the apparently insignificant endobronchial lesion.

Other bronchogenic carcinomas are collar-button in shape, with a partially obstructing endobronchial lesion and a large extra-bronchial component, which may so compress the bronchus proximal to the intrabronchial tumor that tissue for biopsy cannot be reached.

Bronchogenic carcinoma produces no symptoms or findings that may be considered as characteristic of this serious disease; however, it is generally suspected when partial or complete bronchial obstruction is found, or when signs and symptoms of bronchial obstruction recur after having responded temporarily to antibiotic therapy.

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14. DOLL, RICHARD, and HILL, BRADFORD, Ph.D., London, England.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its Annual Assembly in Otolaryngology from September 6 to 11, 1954. The entire week will be devoted to surgical anatomy and cadaver dissection of the head and neck, and histopathology of the ear, nose and throat. The Assembly will be under the direction of Maurice F. Snitman, M.D.

Registration will be limited. For information write to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12, Illinois.

SYMPOSIUM:
BRONCHOGENIC CARCINOMA.
III—TREATMENT.* (SURGICAL TREATMENT)

Richard W. Yore, M.D.,
(By Invitation)
Seattle, Wash.

In this brief discussion of certain aspects of lung cancer I would ask your indulgence in any dogmatism that might arise in the course of this portion of the Symposium for the sake of emphasizing and re-emphasizing features of surgical treatment.

Certain general remarks concerning the treatment of pulmonary malignancy might be appropriate and also interesting. There has been an alarming and disconcerting wave of pessimism, discouraging both patient and physician, that is not helpful in our continuing efforts in improving the results of treatment. Certainly a healthy degree of realism is both necessary and fair, but an unhealthy deplorable onus of despair has begun to creep into discussions about this disease, on the hospital wards, consultation rooms, and hospital residents' quarters. By way of dispelling some of this gloom, one need only remember that the history of the surgical treatment of carcinoma of the lung is just one-half as long as that of surgical therapy of gastric cancer, and yet the over-all results of treatment of these two diseases are the same as measured by the somewhat misleading yardstick—the five-year survival. It would be much more fair and reasonable that the surgical treatment of cancer of the lung should be undertaken with an optimistically expectant attitude.

It has been widely accepted that statistically we are facing a serious and ominous real increase in cancer of the lung. Dr. Evarts Graham has pointed out that in this country and in England and Wales during the last 25 years, there has been a

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15-fold increase in the incidence of cancer of the lung, whereas in the 15 years between 1930 and 1945 there was an insignificant increase (compared to an increase in the number of people reaching the cancer age) of only 600 deaths from cancer of the stomach. It is not so important, however, that cancer of the lung is more common than gastric cancer, but that every year there is an alarming increased incidence of bronchogenic carcinoma. Dr. Blades, too, in the same discussion, showed that from the Medical Statistics department of the Veterans Administration, where there was a predominant male population of patients, there was a terrifying increase of this disease from 1949 through 1951.

A point that Dr. Morgan has previously mentioned, and which I would like to re-emphasize, is this: no other visceral cancer offers an equal opportunity for detection in the presymptomatic growth period, because of the ease of detecting abnormal areas of density in the normally radiotranslucent pulmonary parenchyma by simple X-ray examination.

Published and unpublished experiences with cancer of the lung have indicated that life expectancy is short following diagnosis, and that the duration of life has not been influenced greatly by any means other than surgical extirpation of the neoplasm; therefore, we should discuss surgical treatment as the primary and most successful form of therapy, and mentions others as ancillary, palliative, or secondary methods.

Surgical extirpation or resection as the treatment of choice in cancer of the lung has three main operative procedures. *(a)* exploratory thoracotomy, *(b)* pneumonectomy, *(c)* lobectomy. These are fairly well standardized operations and have been improved, modified, and made more radical in their scope during the past 15 or 20 years. Gibbon, in a beautifully presented and analyzed series of cases, reviewed also the results of 17 other series reported in the literature and called attention to the fact that there is a direct relationship between the number of cases explored and the number of patients leaving the hospital alive after having had their cancers removed: or, in another way of expression, a doubled rate of exploration yields a doubled rate of resection, and a similar increase in patients leaving the hospital following removal of

the malignancy. As one examines the various series reported, it becomes apparent that the indications for attempting extirpation are not universally drawn. What, then, might be the indications for thoracotomy? Dr. Morgan has already alluded to these by the presentation of the four cases in which his index of suspicion was high; *(1)* solitary circumscribed nodule, *(2)* hemoptysis, *(3)* "pneumonitis," and *(4)* the patient with a localized wheeze. Any patient with an undiagnosed, unexplained shadow in the lung, which persists or undergoes slow resolution, and who has all other diagnostic measures negative, should be unhesitatingly explored. Even though it is dangerous to point out contraindications to thoracotomy, since they are rapidly changing and disappearing in recent years, we could briefly classify them as absolute and presumptive contraindications.

Concerning the procedures of pneumonectomy and lobectomy, it must be pointed out that both of these are performed on the basis of one of two fundamental assumptions; namely, that the disease is a localized process and therefore might be eradicated by surgery, and that removal of the lung or lobes might alleviate suffering arising from pain, hemorrhage, or suppuration in uncontrollable cancer, thus making a limited period of survival more comfortable. The important question of whether significantly better results can be achieved if the whole lung and regional nodes are removed or if only a lobe with its nodes is resected, cannot be answered until a much larger series of lobectomy cases has been thoroughly studied and compared with pneumonectomy results. As it stands today, pneumonectomy is the best operation, providing it entails removal of the entire anatomical unit to include adjacent regional lymph nodes. Lobectomy is indicated in the following situations: *1.* Evidence of diminished pulmonary or cardiac reserve (the danger of producing the "respiratory cripple"); *2.* When the carcinoma has extended beyond surgical bounds, but when the bulk of necrotic, suppurative, infective tumor could be removed by lobectomy with hope of relieving the symptoms. *3.* For profound incapacitating and lingering hemorrhage, for severe pain secondary to chest wall invasion, and for localized abscess and resultant sepsis, even though there are evidences of distant slow-growing metastases.

As for "palliative" resections, or resections done in the case of a hopelessly incurable situation, this has been a controversial matter for a number of years. Generally, I believe that removal of necrotic, infected lung tissue reduces the amount of sepsis and continued inflammatory exudate, which the patient would otherwise have to miserably endure. Dr. Ochsner's large series of over 1000 cases showed that 10 per cent of the palliative extirpations survived more than three years, whereas only three of the 200 non-resected cases were alive at the end of two years. Mortality of poor risk palliative resection cases has been reduced from 60 to 20 per cent. This ever decreasing mortality rate, coupled with the personal observation that generally palliative extirpation favorably influences survival time, as well as the patient's ultimate comfort, has influenced many surgeons to recommend and practice this type of surgical approach to a difficult problem.

With an encouraging reduction in operative deaths, which is the result of improved surgical technique, improved anesthesia, blood replacement therapy, and general adoption of antibiotics, there were similarly encouraging advances made in extending the limits of resectability, as by this extension we were able to salvage more patients from the distressing "open and close" category.

Dr. John Gibbon, Jr., made a tremendous contribution in bringing to our attention the technique of intrapericardial ligation of hilar vessels, which increased by 50 per cent the number of successfully resected cases otherwise felt to be non-resectable because of extensive precarious involvement of the large pulmonary arteries, and/or veins. Partial resection of the chest wall, followed by stable reconstruction with tantalum mesh, has increased the number of successfully extirpated peripherally located tumors removed *en bloc* with lobe and segment of chest wall. Similar techniques have been applied to favorable lesions involving a diaphragmatic leaf to still further increase successful radical management of previously considered hopeless cases. It is now commonplace to remove large segments of the pericardium when this layer seems involved by contiguity. In carefully selected cases with an otherwise good prognosis, lesions extending high on one

side of the trachea may be "shaved off" beyond microscopic tumor, and the patients survive three to five years comfortably.

Recently, cancers extending to involve the carina have been resected by removal of the lung, resection of the carina, and anastomosis of the contralateral bronchus to the transected trachea by one of several possible methods. Emphasis on techniques of more extensive and complete *en bloc* regional node excision by Cahan, Higginson (of this city) and others, are too recently tried and as yet seldom used in a general way to evaluate their apparent advantage in increasing the survival rates. These workers and their modifications certainly demonstrate a more definitive anatomically organized radical attack on the regional nodes.

In spite of these advancements in techniques, patient care, and improved anaesthetics, the over-all results in treatment of cancer of the lung are quite depressing. In round figures, Dr. De Bakey pointed out that in a given group of 25 cases of pulmonary neoplasm, about 15 of the 25 would meet the criteria of operability and thereby be explored. Approximately nine, or one-third, would prove resectable; about seven patients, or one-fourth of the original number, would survive the operation, and only two, or approximately 8 per cent, would survive five years or longer. To put the situation in even more simple terms, Ochsner stated that out of every three patients with a reasonably certain diagnosis of lung cancer, one would be eliminated because of obviously inoperable growth, one would have non-resectable growth (or possibly refuse surgery), and one would actually have a resectable lesion. In general, in most series a sufficient period of time has not yet elapsed to give an indication of the relationship between the number of patients having had cancer of the lung that leave the hospital following removal of the lesion and the number that survive five years *or more* without evidence of residual cancer. At present there is no indication that there will not be a direct relationship between the two groups. In Gibbon's series most recently published, the analysis covered 532 consecutive proven cases, and the interesting results were as follows: *(a)* 22 per cent operative mortality in patients in whom the cancer was extripated; *(b)* the five-

year survival rate was 22 per cent of patients who had cancer removed, and 9 per cent of the entire series. He did not practice palliative resections. In his series an increase in exploration rate of 17 per cent above the next highest rate reported was rewarded by only a 4 per cent increase in the rate of extirpation and a 2 per cent increase in patients leaving the hospital following removal of their cancer.

What are the reasons for such disappointing results in spite of more progressive attacks on the disease? Dr. Morgan mentioned the disease, patient and physician factors responsible for delay or interference with successful therapy. Dr. John Jones in reviewing and analyzing his series of 633 cases brought out that the recent increase in more far-advanced cases is due to increased delay between time of first patient-physician contact and time of surgery recommended. He proposed that increased employment of antibiotics is responsible since they are used to treat recurring lung infections, while the carcinoma remains unsuspected as the cause of the "pneumonitis." In our experience this has been all too true, and notables in this field such as Dr. E. A. Graham and Dr. Robert Janes have preached against incorrect usage of the antibiotics with a low index of suspicion. One other possible reason for patient and/or doctor delay is the false sense of security produced by patient saying to himself "Oh, I can't have cancer of the lung because my chest X-ray a year ago was entirely negative." We must not allow this common misconception concerning chest X-ray findings in early lesion detection.

Just as Dr. Morgan made plea to those of you who do bronchoscopy, so would I like to enlarge upon your importance as a bronchoscopist in the management of suspected cancer of the lung. The bronchoscopist can be of inestimable value to the thoracic surgeon when he provides him with information about fixation, the carina and its configuration and mobility, distortion or concentric narrowing of bronchial lumina as well as bronchial wall biopsy to pick up the submucosal induration of the wall. He should be well and accurately versed in segmental anatomy, not only from the "inside," but also from the "outside"; *i.e.* by X-ray localization, so that "blind" biopsies can be taken from the correct segment involved in

the X-ray process. He should develop a strong intellectual curiosity about all and any abnormalities, and bold enough with biopsy forceps to try to confirm microscopically his observation of abnormal findings by a properly gauged biopsy.

In summary, therefore, we have discussed various phases of surgical treatment of carcinoma of the lung, beginning with general remarks about the problem and the alarming increase in incidence, emphasizing the importance of early thoracotomy, mentioning the various forms of surgical treatment and their results. I am sure that it has become most clear to all of you that of all the factors working for the improved results of treatment, the most important is and always will be *suspicion.*

UNIVERSITY OF ILLINOIS COLLEGE OF MEDICINE.

The Department of Otolaryngology, University of Illinois College of Medicine, announces its basic science course in otolaryngology offered by its affiliated hospitals. This combined postgraduate course and residency will begin its 1954-55 session on July 1, 1954. Other openings occur throughout the year. Residencies are available at either the Research and Educational Hospital or the Illinois Eye and Ear Infirmary, or a continuation of the training program may be arranged for the Veterans Administration Hospital at Hines.

A stipend is offered on the following basis: First year residency, $1320 annually; second year residency, $1620 annually; third year residency, $1920 annually.

Application forms are available on request to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12.

OTORHINOLOGIC ASPECTS OF HAND-SCHULLER-CHRISTIAN'S DISEASE.*

J. JULIAN CHISOLM, M.D.,
Baltimore, Md.

Every otolaryngologist should be acquainted with the symptomatology of the clinical syndrome known as Hand-Schüller-Christian's disease. In children it may simulate chronic infections involving the temporal bone and neoplasms or cysts of the paranasal sinuses. It has received far more attention in the pediatric textbooks than in those of our specialty.

Hand, in 1893,[1] described the autopsy findings in a case, but he considered it a peculiar type of tuberculosis. Schüller, in 1915,[2] pointed out the characteristic radiological appearance of the skull defects. Christian, in 1919,[3] published his "Defects in the Membranous Bones Exophthalmos and Diabetes Insipidus" and, thereafter, the name Hand-Schüller-Christian's disease was used to describe cases exhibiting this triad. In 1928, Rowland ascribed the syndrome to a type of xanthomatosis, and for some years thereafter, Hand-Schüller-Christian's syndrome appeared in the pediatric textbooks in the group of disases of abnormal lipid metabolism of obscure origin; however, since 1940,[4] further pathological studies and clinical observations have resulted in the grouping of Hand-Schüller-Christian's disease with two other closely related conditions—Letterer Siwe's disease and eosinophylic granuloma. The three are now generally referred to as variant types of systemic reticuloendotheliosis,[5] the essential change being a proliferative response of embryonal reticular cells of the body to a stimulating and as yet unknown agent. The clinical pictures differ so strikingly that the three are usually classified as separate disease entities, but the pathologic process shows the same basic pattern in each. Goodhill[6] suggests "Histiocytic granuloma" as a better all-inclusive term than systemic reticuloendotheliosis, and reports 18 cases, some of which were

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followed over a period of years and showed transition from one type to another.

Letterer-Siwe's disease, the acute, rapidly fatal form, usually occurs in infants or before the age of two years. The clinical picture includes general lymphadenopathy, progressive anemia, purpura, fever, skin rash, cachexia and occasional enlargement of the liver and spleen, and skeletal involvement. The lesions are widespread and microscopic examination of them reveals numerous reticular cells, histiocytes, a few giant cells and occasional foam cells, plasma cells and eosinophiles. Siwe suggested that the disease be called "diffuse reticuloendotheliosis." As a rule X-ray therapy does not influence the course of the disease, and death usually occurs within a few weeks or months.

Eosinophilic granuloma, the chronic type of reticuloendotheliosis, was first described by Fraser in 1935.[7] He called it eosinophilic osteomyelitis and considered it a variant of lipoid granulomatosis. The clinical picture is that of a solitary benign tumor of the bone. Under the microscope the tumor is found to be made up chiefly of histiocytes, eosinophiles and giant cells. It may occur in the long bones or the skull and responds equally well to surgical removal or deep X-ray therapy; however, recurrence in other parts of the skeletal system is not uncommon. The same patient may later develop diabetes insipidus or multiple lesions of the Hand-Schüller-Christian type. Love and Fashena[8] reported the case of a child who developed a solitary tumor of the right mandible, diagnosed as eosinophilic granuloma, at the age of 19 months, then three years later diabetes insipidus, and four years later general lymphadenopathy.

The third or subacute type of systemic reticuloendotheliosis is Hand-Schüller-Christian's disease. It has also been called chronic idiopathic xanthomatosis or lipoid granulomatosis. The clinical triad usually associated with it consists of: *1.* X-ray defects in the membranous bones: skull, pelvic girdle and scapulae; *2.* diabetes insipidus; and *3.* exophthalmos. The symptomatology will depend upon the location of the bony lesions. The bones of the orbit and the sella turcica seem to be sites of predilection, which accounts for the frequency of

proptosis and diabetes insipidus. When the first symptoms are referable to the temporal bone or the superior maxilla, the case is more likely to fall into the hands of the otolaryngologist at an early stage. He should be on the alert for unusual cases of this type, and X-ray examinations should include the other membranous bones. The films may reveal lesions elsewhere in the skull or in the pelvic girdle or the scapulae, which have as yet given no symptoms. The X-ray changes characteristic of Hand-Schüller-Christian's disease have been likened in appearance to lakes on a map or moth-eaten holes of varying size.

Histologically the lesions are identical in all areas involved and exhibit a peculiar type of inflammatory reaction with eosinophilic and polymorphonuclear leucocytes, a proliferation of reticulum cells, histiocytes, foam cells and giant cells. The foam or xanthoma cells, large mononuclears with lipid vacuoles, are filled with cholesterol. They predominate in the lesions. Their presence accounts for the fact that Hand-Schüller-Christian's disease was originally grouped with xanthomatoses. The proliferative xanthoma-like masses originating in the neighboring bone structures may invade the middle ear cavity, the external auditory canal, the orbit, the antrum, or the pituitary. The characteristic diabetes insipidus is caused by invasion of the pituitary from the sella turcica. In the middle ear, or external auditory canal, the masses may be easily mistaken for aural polypi or cholesteatoma. In the orbit or antrum they may simulate tumors. Goodhill[6] reports the temporal bone findings in a case which came to autopsy and, although there was extensive involvement of the mastoid and petrous pyramid, the bony capsule of the labyrinth resisted invasion, and the cochlea and vestibular apparatus were essentially normal. Eggston and Wolff's textbook of Histopathology[9] pictures a histological section of a temporal bone, which shows the characteristic microscopic appearance. Although the xanthoma cells are rich in cholesterol, the blood level of cholesterol is always normal.

The onset of Hand-Schüller-Christian's disease is insidious and usually between the ages of two and six; however, it sometimes develops at a later age. Troxler and Niemetz[10]

report a case in an adult of 35, with diabetes insipidus, generalized xanthomatosis affecting the vertebrae, pelvis and femora, and bilateral lung involvement and pneumothorax. The disease had given its first symptom at the age of 20, when all the teeth began to loosen as the result of changes in the alveolar process. The presenting symptom depends on the location of the first bone lesion—it may be proptosis, diabetes insipidus, a swelling of the cheek suggestive of a malignant tumor or a congenital cyst, a swelling of the mandible with loosening of the lower teeth, or a discharging ear without preceding pain or acute respiratory infection. There may be a low-grade fever, but pain is seldom a symptom. When the lungs are involved there is cough and cyanosis, and X-rays of the chest may reveal changes suggestive of miliary tuberculosis, atelectasis, or cavity formation. Skin lesions are occasionally present and they resemble seborrheic dermatitis, but there is no itching. X-ray changes in the bones must be differentiated from those occurring in multiple myeloma, solitary bone tumors, malignant metastases and chloroma.

The prognosis is uncertain. The bone lesions are usually multiple, tend to resolve spontaneously and to be replaced with fibrous tissue and new bone, only to recur in other areas over a period of years. X-ray therapy causes rapid resolution. The diabetes insipidus can be controlled satisfactorily by pitressin. When secondary infection occurs, as frequently happens in the ear, an appropriate antibiotic is helpful. As the cause or stimulating agent is unknown there is no specific therapy. Death may occur from visceral or brain lesions or a transition to the Letterer-Siwe type; however, after a period of years the condition may become quiescent. If the pituitary has been involved the diabetes insipidus requires continuous treatment, the child's growth is arrested and dwarfism and hypogenitalism become evident as the patient matures. The writer recently had the opportunity of seeing a boy, now 15, who developed Hand-Schüller-Christian's disease at the age of three, the first bony lesion being in the mandible. He then had recurrent involvement of the membranous bones over a period of six years, though no lesions appeared in the temporal bones. He is now an infantile dwarf with diabetes insipidus. It is interesting that while X-rays

showed almost complete destruction of the sella turcica, in the early stages of the disease, it has now regenerated. Recent films reveal a well formed but curiously shaped sella with elongated clinoid processes.

CASE REPORTS.

Case 1. G. H., white, age 27 months, first seen by me June 22, 1949. Referred by Dr. Harriet Guild of the Pediatric Department, for a routine check-up.

Personal History: No ear infections. In the summer of 1948 he developed a tumor of the left orbit—X-rays in the Pediatric Department August 1, 1948, resulted in a diagnosis of Hand-Schüller-Christian's disease. The proptosis disappeared completely after X-ray therapy, and there has been no recurrence.

Examination: June 22, 1949, revealed no abnormality in the ears, nose and throat. X-rays of the membranous bones revealed no new lesions. He was next seen for a check-up November 4, 1953, then six and one-half years old and large for his age. In the spring of 1951, he had developed a lesion in the pelvic bone, which cleared up with X-ray therapy; otherwise, there have been no further manifestations of Hand-Schüller Christian's disease.

At this time, he had a cough due to infection of the adenoids. The right antrum was rather dark on transillumination. X-ray of the sinuses revealed some marginal clouding of the right antrum, but no bone lesions in the superior maxilla. X-rays of the skull and pelvis were negative. This case illustrates a mild type of the syndrome with only two bone lesions: one in the skull, and one in the pelvis, over a period of more than five years. He has not had diabetes insipidus.

Case 2. J. F., female, age three and one-half years. First seen by me June 30, 1951. Referred by Dr. Harriet Guild of the Pediatric Department.

Complaint: Discharge from the left ear, impairment of hearing.

Past History: Bilateral otitis media at 18 months with several recurrences since then. About February 1, 1951, mother noticed a soft painless swelling in the scalp of the occiput just above the hairline, which was thought to be a cyst under the skin.

Present Illness: On March 1 mother took the child to her local pediatrician because she was very irritable, though there were no specific complaints. He noted a swelling in the roof of the left external auditory canal, suggestive of a localized infection in the skin. Several weeks later, a polyp appeared in the left canal, and it was removed by an otologist. A second polyp developed within a few weeks, and it was removed under general anesthesia May 24, 1951. After this the left ear began to discharge, and the hearing in both ears seemed impaired.

Examination: June 30, 1951. Tonsils and adenoids enlarged. *Ears:* right drum intact, opaque; left canal filled with foul smelling purulent discharge and a large, pale yellowish, polypoid mass arising from the roof of the bony canal; the left drum could not be seen. There was obvious impairment for test words in both ears. The skin over the left mastoid and the left post-auricular fold exhibited eczematous changes. There was a discharging sinus tract in the occipital region.

Culture: left ear: staphylococcus albus, resistant to penicillin, sensitive to aureomycin and terramycin. X-rays taken June 29, 1951, showed a very large area of destruction in the occipital bone and multiple areas in both petrous pyramids (see Fig. 1). There was also extensive destruction in the left temporal bone.

Fig. 1 (Case 2). X-ray of base of skull, taken June 29, 1951, showing large area of destruction in the occipital bone and multiple areas in both petrous pyramids.

The child was started on aureomycin and a course of X-ray therapy. She returned July 30, 1951, after having had nine X-ray treatments over a period of three weeks. The left ear was clean and dry, the drum intact, the polyp had completely disappeared and a small defect could be seen in the roof of the bony canal. The discharging sinus tract in the occiput had healed. Repeat X-ray films on July 31, 1951, showed no change as yet in the appearance of the left temporal and occipital bones (see Fig. 2). No other lesions had appeared. Since then, the child has been brought back to Baltimore repeatedly, and a succession of other bone lesions have developed and then regressed under X-ray therapy.

A brief resume of events follows: December 17, 1951. *Left ear:* normal, defect in bony canal healed. *Right ear:* skin over the mastoid

Fig. 2 (Case 2). Lateral X-ray of skull taken July 31, 1951, showing extensive areas of destruction in the left temporal and occipital bones.

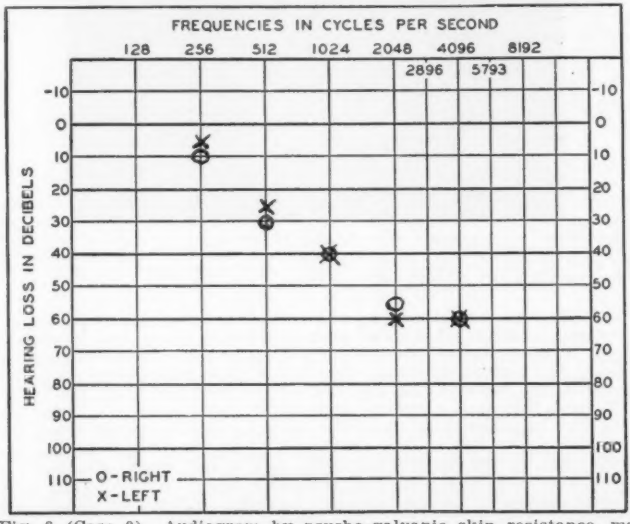

Fig. 3 (Case 2). Audiogram by psycho-galvanic skin resistance, made February 5, 1952. It reveals a bilateral nerve type of hearing loss.

eczematous; large cholesteatoma-like mass filling the canal and arising from the roof. It was easily removed with a small curette, had a foul odor, and a culture made from it was reported staphylococcus albus. At its site of attachment, there was a bony defect in the roof of the canal, filled with pale granulation tissue. X-rays revealed changes in the right temporal bone similar to those in the left, described above. A new lesion has appeared in the occipital bone and the child has just developed diabetes insipidus.

Fig 4 (Case 2). Anterior-posterior view of skull made May 29, 1952, showing marked clouding of the right maxillary sinus.

Another course of X-ray therapy was started. February 5, 1952: Both canals look normal, right drum opaque and thickened, left drum normal. Psychogalvanic skin resistance hearing tests verify a subjective audiogram done October 11, 1951. There is a marked bilateral impairment of hearing of the nerve type (see Fig. 3). Diabetes insipidus well controlled with pitressin. New lesions have appeared in the pelvic bones.

May 22, 1952: Ears look normal. New lesions have appeared in the skull. The face looks normal, but anterior-posterior X-rays of the skull taken May 29, 1952, show marked clouding of the right antrum (see Fig. 4). November 13, 1952: Swelling of the right cheek first noted two weeks ago. Nose looks normal. There is a definite protrusion of

the right eye and a firm swelling of the right cheek, which might easily be mistaken for a malignant tumor of the right antrum.

February 13, 1953: Swelling of the right cheek subsided. New lesions in the occiput and mandible. April 10, 1953: X-rays of the skull show extent of repair of bone changes seen in Fig. 2 (see Fig. 5). October 14, 1953: Audiogram (subjective) made today shows a drop of 10 db in both ears for C256, a drop of 20 db for C4096 in the right ear and an apparent improvement of 30 db for C4096 in the left ear.

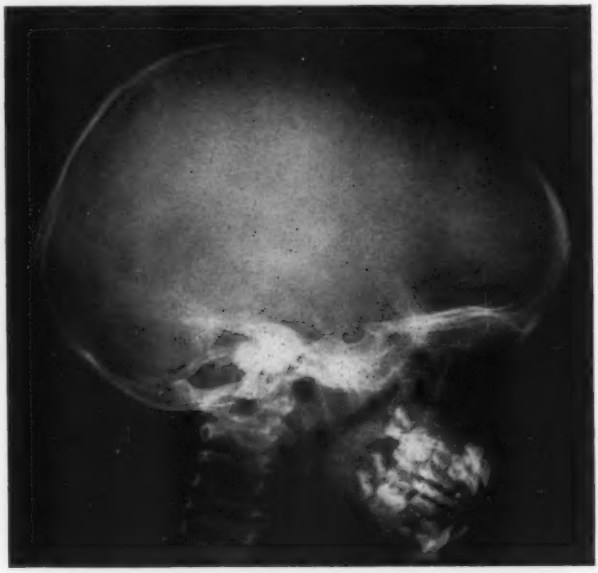

Fig. 5 (Case 2). Lateral X-rays of skull taken April 10, 1953, showing extent of repair of bone changes seen in Fig. 2. There is also a new area in the left parietal bone.

The proptosis of the right eye recurred in the spring, began to regress August 20, 1953, and has varied in degree since then. X-rays show a defect in the supra-orbital ridge on the right and a new lesion in the posterior wall of the left orbit. Lesions in the right parietal region and the left pubis are healing.

Case 3: M. L., female, age nine years. Admitted to the Johns Hopkins Hospital July 27, 1953, as a patient of Dr. Walter E. Loch. *Complaint:* Impairment of hearing.

Family History: Allergy on both sides. *Past History:* Infantile eczema. Asthma since six months of age. No trouble with ears until March, 1950, when at the age of five years nine months, she developed purulent discharge from the left ear, accompanied by post-auricular swelling.

The left mastoid was opened, and a month later the wound healed. In June, 1950, after swimming, both ears began to discharge, hearing

became impaired and she complained of dizziness. In August, 1950, a bilateral mastoidectomy was done. Both ears became dry, but the hearing remained poor. In June, 1952, she developed diabetes insipidus. In December, 1952, both ears began to drain again and the discharge from the left ear persisted in spite of antibotic therapy.

She was brought to Baltimore for the first time in July, 1953. Examination on admission July 27, 1953: Upper respiratory tract normal. Left ear: draining mastoid fistula, blood tinged purulent discharge in the external canal and a mass adherent to the anterior wall in the midportion, hiding the drum. Hearing: Left ear nil: right ear profound loss X-rays showed defects in both petrous bones, no demonstrable lesions elsewhere in the skull and a questionable lesion in the right pubis.

On the basis of the history of diabetes insipidus, Hand-Schüller-Christian's disease was suspected, in spite of the absence of characteristic X-ray findings. Radical mastoidectomy, left, July 30, 1953 (Dr. Loch). The cavity was filled with grayish-yellow tissue (frozen section diagnosed acute inflammatory reaction). The lateral sinus and the dura of the posterior and middle fossae were widely exposed and covered with granulations. A large labyrinth sequestrum was removed, exposing the internal carotid artery. The middle ear was filled with granulations and yellowish tissue; no ossicles could be demonstrated. The operator noted that the gross appearance confirmed the suspicion of Hand-Schüller-Christian's disease.

Pathological report (labyrinth sequestrum): Acute and chronic inflammatory reaction with many eosinophiles, large mononuclear cells and necrosis, marrow spaces infiltrated with small mononuclears, eosinophiles and giant cells. Diagnosis: Hand-Schüller-Christian's disease. X-ray therapy was begun August 10, 1953. Patient was last seen Novembers 29, 1953. Ear dry. X-rays show continued healing of defects in the petrous bones and no new bone lesions elsewhere. Has gained nine pounds in weight.

CONCLUSIONS.

1. Hand-Schüller-Christian's disease may simulate middle ear or mastoid suppuration in children. The sudden appearance of an aural polyp in a previously normal ear should make one suspicious. X-ray examinations of suspected cases should include the entire skull and other membranous bones, as well as the temporal bones. A history of diabetes insipidus is a valuable clue.

2. If the case is diagnosed early, X-ray therapy plus an appropriate antibiotic can result in rapid resolution (Case 2); however, multiple lesions in the petrous pyramids may lead to a permanent hearing loss of the nerve type, probably due to encroachment on the internal auditory canal (see audiogram, Fig. 3). Autopsy reports reveal that the capsule of the cochlea is resistant to invasion, yet in Case 3, repeated and undiagnosed lesions in the temporal bone resulted in a sequestration of the labyrinth and a total loss of hearing.

3. Hand-Schüller-Christian's disease may simulate tumors of the antrum as well as orbital tumors. X-ray of the sinuses alone may fail to reveal the true nature of the disease.

4. The prognosis is uncertain. Although the bone lesions respond well to X-ray therapy, they tend to recur in all the membranous bones over a period of years. Case 2 has had innumerable lesions in the past three years. Cases with pituitary involvement and diabetes insipidus, if they survive the early years, are likely to be infantile dwarfs.

5. The bone lesions tend to resolve so completely that X-rays several months or years later may reveal little or no trace of them; thus, in a case of diabetes insipidus, due originally to destructive lesions in the sella turcica, X-ray films after several years may picture a fairly normal sella.

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FURTHER EXPERIENCES WITH DIHYDROGENATED ERGOT ALKALOIDS IN CERTAIN OTOLARYNGOLOGIC DISORDERS.

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We recently reported the results obtained in the treatment of 116 cases, with sublingual Hydergine (R), a sympathetic inhibitor.[1] Since the preliminary report we have continued using this mixture of dihydrogenated ergot alkaloids, and our purpose in this presentation is to report our results in an additional 210 cases (see Table I).

TABLE I—TYPES OF CASES.

Vertigo	77
Hypertensive epistaxis	10
Dryness of the mouth	30
Glossodynia	19
Post-nasal discharge	58
Atrophic rhinitis and nasopharyngitis	17
Miscellaneous	9
Total this series	210
Previous series	116
Total clinical evaluation	326

Just as was emphasized in our original communication, we are aware that the clinical behavior of many of the disorders treated is erratic, and spontaneous regression may be as sudden and complete as it was in many of the cases we report. In order to eliminate this pitfall, we are at present undertaking a blind placebo study at Charity Hospital in New Orleans. Our intention is to publish the results of this clinical evaluation at a later date.

PHARMACOLOGY.

The best-known action of the ergotamine and ergotoxine drugs is vasoconstriction; not so well-known is the fact that they also have a sympathetic blocking action. Recently,[2] as a

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result of hydrogenation of the ergotoxine group, it has been possible to eliminate the vasocontrictor action so that the sympathetic blocking action becomes predominant.

Other actions ascribed to Hydergine are:

1. Depression of the central vasomotor centers with resultant vasodilatation and in some cases, a fall in blood pressure.[3]

2. Peripheral vasodilatation.[4]

3. Central inhibition of pressor-receptor reflexes which bring about compensatory vasocontriction and arterial blood pressure rise.[5]

4. Centrally induced bradycardia; this is rather unusual among sympathetic blocking agents.[6]

5. Central sedative action; also augmentation of barbiturate action.[7]

6. An adrenosympathicolytic effect.[8]

7. A possibility that oxygen utilization might be increased.[9]

8. Decrease in vascular distensibility, increase in venous pressure and increasing prominence of arterial pulsations.[10]

Toxic Manifestations. In this portion of our study, only two individuals reported untoward reactions: both were minor and subsided promptly when the drug was discontinued.

RATIONALE OF TREATMENT WITH HYDERGINE.

Vertigo. The most widely-held present-day concept of the pathologic physiology of many cases of labyrinthine vertigo is that there is arteriolar spasm with dilatation of the capillary-venule network distal to the spastic segment. It is felt that in the distended loop there is sludging of blood, liberation of toxic metabolites, among which is histamine; anoxia of the blood vessel walls, and increased capillary permeability which leads to tissue edema and possibly increased accumulation of endolymph.

The effectiveness of Hydergine in these cases may be the result of a combination of its pharmacodynamic actions. The vascular spasm is probably relieved by the peripheral vasodilatation. Once relieved, the spastic effect is probably prevented from recurring by depression of the central vasomotor center; further, the central sedative effect undoubtedly serves to allay some of the psychosomatic element usually present in

these conditions. Other phenomena of importance may be the fact that during Hydergine therapy a decrease in vascular distensibility, increase in venous pressure, and increasing prominence of arterial pulsations have been noted; this would undoubtedly serve to relieve the congested state of the part. Finally if, indeed, Hydergine does increase oxygen absorption or utilization, it may help to reestablish enzymatic and other chemical homeostasis in the affected tissues.

Epistaxis of Hypertensive Origin. In the management of epistaxis of hypertensive origin, more than merely stopping the bleeding is desirable; attempts to lower the blood pressure should also be made.

Most cases of hypertension are now believed to be of psychosomatic origin; those which are not, undoubtedly have developed some psychosomatic overlay, this being particularly true in the hypertensive epistaxis. The psychosomatic disturbance is said to be mediated through the autonomic nervous system in the form of relative sympathetic preponderance, with resultant generalized vasconstriction of the vascular bed. In the kidneys, vasconstriction causes elaboration of long-acting pressor substances into the circulation.

The use of Hydergine in these cases is predicated on its ability to depress the sympathetic neurogenic influences, to provide peripheral vasodilatation, to provide central sedation (in addition to augmenting barbiturate action), and to lower the blood pressure; all these effects are obtained with the production of none of the signs of vasomotor collapse.

Dryness of the Mouth. Salivary glands have both a sympathetic and parasympathetic nerve supply. They differ from other effector organs, however, in that both elements of the autonomic nervous system are cholinergic.[11] A decrease in salivary output, therefore, could be due either to a hypofunctioning autonomic nervous system or to hypofunctioning acini of the glands (possible, in some instances, at least, on the basis of a decreased blood supply). The effectiveness of Hydergine may be due to its peripheral vasodilator action, or to the depression of the central vasomotor system. Most cases respond to Hydergine alone, but it may be necessary to use

prostigmin bromide in addition, for its action in destroying choline esterase and thus permitting freer transmission of cholinergic impulses.

Parenthetically, we might mention that we have used hydergine in a number of cases complaining of the "dry, scratchy feeling" in the nose, nasopharynx, pharynx, or larynx that often is the harbinger of the common cold or a catarrhal upper respiratory infection. Our reasoning was that if the mucous glands of the area could be stimulated, the condition would be relieved; this proved to be the case in many instances.

Glossodynia. Hydergine was used in patients complaining of painful, burning tongue, on the theory that, in some cases at least, the symptoms might be the result of a circulatory disturbance and, therefore, would be benefited by vasodilatation, the increased blood flow leading to improved nutrition and a "flushing out" of accumulated toxic metabolites. It is interesting to note that some of the vitamins used in the past are potent vasodilators.

POSTNASAL DISCHARGE.

Patients complain of nonpurulent postnasal discharge either *1.* when the amount of mucus is profuse, *e.g.*, as in allergic rhinitis, or *2.* when the mucus is thick and does not move normally, or rolls over in masses or globules. In the latter instance the individual becomes conscious of the thickened mucus and begins to make strenuous efforts to remove it, sometimes going so far as to produce nausea and vomiting.

Our rationale in trying Hydergine in these cases was based upon the hope that blockage of the sympathetic nerve supply would leave the parasympathetic secretory system unopposed, and secondly, that the vasodilatation produced would improve the blood supply and thus re-establish normal viscosity. The object was not to "cure," or abolish, the postnasal discharge; rather, it was to re-establish the normal mucus pattern.

ATROPHIC RHINITIS AND NASOPHARYNGITIS.

There is general agreement that in ozena there is a decrease in the blood supply to the nasal mucosa. Whether this is pri-

mary or secondary to other factors is not known; nevertheless, there have been reports concering the usefulness of vasodilating agents in this condition. It was for this reason that Hydergine was used in these cases.

MISCELLANEOUS CONDITIONS.

We have used Hydergine in several cases of Bell's palsy in an attempt to restore circulatory dynamics in the facial nerve, as it courses through the Fallopian canal of the temporal bone. The rationale of its use here is the same as that for the use of histamine in this condition.

Several cases of cranial nerve neuralgia were administered Hydergine, because it has been suggested that these conditions may be due to ischemia of the nuclei.

DOSAGE.

The medication was administered in the form of sublingual tablets containing equal parts of the three hydrogenated alkaloids of ergotoxine.* The patients were instructed to dissolve one or more tablets under the tongue four to six times daily.

RESULTS.

Vertigo. Sixty-four of 77 cases demonstrated marked to complete remission of symptoms; the remainder either reported no relief or only slight improvement.

Hypertensive Epistaxis. It was our clinical impression that the use of Hydergine in these 11 cases made their management much simpler. Objectively, the decrease in blood pressure was usually dramatic and similar to that recounted in our previous report.

Dryness of the Mouth. The results in this condition were singularly impressive; 27 of the 30 patients reported marked improvement to complete remission; two patients required a combination of Hydergine and prostigmin bromide to obtain complete remission. In one instance the treatment was completely unsuccessful.

Glossodynia. Of the 19 patients with this complaint, 15 reported rapid relief of symptoms, two were moderately improved, and two noted no improvement.

* The Hydergine (P) used in this study was provided by the Sandoz Pharmaceutical Co.

Postnasal Discharge. All but five of the 58 patients who presented themselves with a thick, tenacious, mucoid postnasal discharge reported marked improvement following a course of Hydergine. Most of these individuals displayed a mild anxiety state, or smoked a considerable number of cigarettes each day.

Atrophic Rhinitis and Nasopharyngitis. Thirteen of the 17 cases with this complaint demonstrated improvement, but relapsed to their former state when the medication was discontinued. There was no noticeable improvement in four cases.

Bell's Palsy. Six patients with unilateral Bell's palsy were treated with Hydergine. Though difficult to evaluate clinically, it was our impression that resolution was speeded.

Cranial Nerve Neuralgia. Three patients with cranial nerve neuralgias were included in the series; two patients showed no improvement. In one individual the attacks seemed to be of a milder nature. This may have been coincidental.

SUMMARY.

1. A report is made of further results of a clinical evaluation of the use of a new adrenergic blocking agent, Hydergine, in several otolaryngologic disorders. Included among these disorders are: vertigo, hypertensive epistaxis, dryness of the mouth, glossodynia, postnasal discharge, atrophic rhinitis and nasopharyngitis, and Bell's palsy.

2. The pharmacology of the dihydrogenated ergot alkaloids is again reviewed.

3. The rationale for the use of hydergine in the disorders considered is presented.

4. The results obtained roughly approximate those of the first clinical evaluation. This leads us to conclude that Hydergine is a useful addition to our therapeutic armamentarium.

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AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Registrations must be completed before August 15. Detailed information and application forms may be secured from Dr. William L. Benedict, the executive secretary-treasurer of the Academy, 100 First Avenue Building, Rochester, Minnesota.

SURGICAL TREATMENT OF THE FRONTAL SINUS BY THE EXTERNAL ROUTE.

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and
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1. A study of the surgical treatment of chronic frontal sinusitis shows early two fundamental and divergent points of view, one directed to the obliteration of the sinus and the other to its conservation with improved intranasal drainage.

The earliest forms of surgery were reserved for the cure of necrosis of the walls and of fistulas.

Runge[1] of Germany, conceived the idea of obliterating the sinus cavity by opening or removing the anterior wall, and treating the tissues with a caustic ointment of nitrate of silver, in an endeavor to unite the soft tissues and mucous membrane lining the bone. Thus, was initiated the idea of the elimination of the sinus cavity.

The majority of surgeons, however, have devoted themselves to the establishment of an efficient and permanent communication between the sinus and the nose.

This paper is based on the conception of the obliteration of the sinus and the closing of the fronto-nasal duct.

This concept is shared with R. R. Woods[2] of Dublin, whose whose opinion is quoted, "Teaching has made us all believe that to succeed in frontal sinus operations, a patent frontal duct must be maintained. I am suggesting that the teaching is quite wrong. For success in this operation the frontal duct must be closed."

2. In general, surgical treatment by the external route is indicated in the following cases:

1. Simple or complicated infections in which medical or surgical treatment has failed to cure or improve the patient's condition.

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Simple cases can be divided into those showing

a. Insufficient natural drainage.

b. Irreversible changes in the mucous membrane.

c. Pyocele.

Complicated cases may be divided into those of

d. Intracraneal complications.

e. Cranial, osteomyelitis of the vault.

f. Orbital.

g. General.

2. Mucocele.

3. Osteomas and other rare tumors of pseudotumoral granulomas.

4. Sinus troubles due to irreversible allergic conditions which block the fronto-ethmoidal infundibulum, provoking pain which does not cede to ordinary medical treatment.

5. As a means of intracranial access to the roof of the ethmoid and the cribriform plate (olfactory fossa) in cases of fissure of the ethmoid with a resultant cranial hydrorrhea.

6. Pneumocraneum of frontal sinus origin.

7. Trauma of the frontal sinus.

Speaking in general terms of the infections of the frontal sinus (which form the majority of the surgical cases), the following procedures may be cited as classical.

1. Ethmoidectomy with removal of the fronto-ethmoidal wall.

2. Opening of the frontal sinus by resection of the

a. Inferior wall. Jansen, Ritter.[3]

b. Partial removal of the inferior wall. Uffenorde-Seiffert-Lynch.[4]

c. Partial removal of the frontal wall. Ogston.[5]

d. Entire removal of the anterior wall. Kuhnt.[6]

e. Total removal of the inferior wall and part of the anterior (Killian-Taptas[7]), etc.

f. Total removal of the inferior wall and partial removal of the anterior, but respecting the integrity of the ostium of the frontal sinus (Walsh).

g. Total removal of the anterior and inferior walls (Riedel[8]).

h. Removal of anterior and posterior walls of the frontal sinus leaving the inferior wall and the fronto-nasal duct or else packing the latter with gelatin sponge soaked in fibrin. Opening of the anterior ethmoidal cells or exceptionally the posterior. The sinus is packed post-operatively with gauze to encourage granulations from the dura and bone to bring about gradual obliteration of the cavity (Mosher[9]).

i. R. R. Woods[10] of Dublin takes away the inferior wall of the frontal sinus and meticulously removes the mucous membrane of the cavity and of the fronto-nasal duct, achieving eventual obliteration of the sinus and duct.

j. Finally we come to the osteo-plastic technique or temporary opening of the anterior wall which was first practiced in 1894 by Brieger and Schönborn,[11] modified by Bergara and more recently by Tato of Buenos Aires.

All other procedures represent modifications of the foregoing list. In the United States the Lynch operation and in England the Howarth operation have many followers.

3. Treatment through the surgical opening of the mucous membrane of the sinus.

a. Total extirpation.

b. Partial extirpation.

c. Conservation of the mucous membrane.

4. Establishment of a permanent fronto-ethmo-nasal communication by

a. Simple enlargement.

b. Flaps using nasal mucous membrane.

c. Free grafts of skin.

d. Tubes—Rubber, Acrylic, Tantalum.

In spite of powerful antibiotics, meticulous care is required in the postoperative treatment as the cavity easily becomes infected as the duct becomes narrowed. In association with Bergaglio, Tato has evolved from the past, old technique, which consists of the occlusion of the sinus cavity and then filling it with fat. This practice of using fat inserts was suggested by our experience in dealing successfully with operative cavities in ear surgery.[14-26]

Riedel tried a technique with the same object, but the bad esthetic results relegated the operation to those severe cases with complications, and finally the intervention was not always successful.

Hajek tried to obliterate the cavity by granulation tissue formation, as Mosher recommends. Deformities and occasional failures followed Hajek's operation.

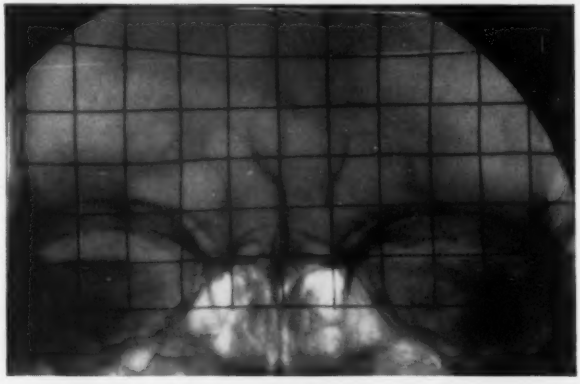

Fig. 1. Radiograph with application of a wire grille, with squares of 1 c.m., in order to determine precisely the limits and outlines of the sinus cavity.

Tato has used the insertion of various materials into the sinus with good results, and his method is the following:

1. Opening: a. Total removal of the inferior wall of the frontal sinus when the sinus is not high although it may be broad and deep. If it is high, temporary resection of

Fig. 2.

the anterior wall by formation of an osteo-plastic flap is the procedure of choice. In cases of bilateral sinusitis, both sinuses may be dealt with from one side by complete removal of the intersinal wall (see Fig. 12); however, in such cases we do a total removal of the inferior wall and the antero-internal portion of the anterior wall below the esthetic eminence.

b. Complete and meticulous removal of the mucous membrane of the sinus, respecting that of the infundibulum.

2. Closure of the communication with the nose:—This is done by separating the mucous membrane from the canal wall sufficiently to form a cuff which is then inverted downwards (see Fig. 9). In case of doubt about closure, a piece of fascia lata or a flap of periosteum from the neighboring bone may be inserted with fat placed above it (see Fig. 10). If the fatty tissue has some aponeurosis attached to it, this latter may be introduced into the infundibulum and suffices to close the opening without anything further being done.

Fig. 3.

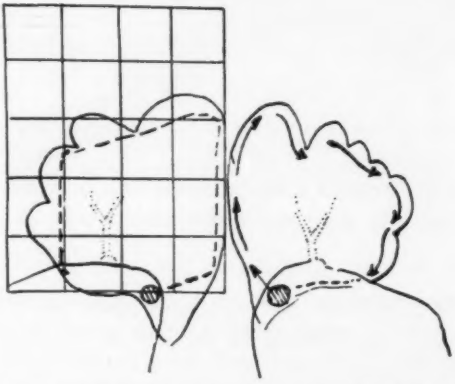

Fig. 4. A diagram to show the importance of the grille to guide the surgeon in the section of the periosteum close to the periphery of the sinus, exposing the bare bone for a width of 5 mm., in preparation for the special burr, which follows the path mapped out by the grille and shown by the arrows. The diagram also shows the point of election for the entry of the burr inferiorly. The dotted line shows the site of the fracture.

3. We do an ethmoidectomy if it is necessary, practicing the transmaxillary route operation (Ermiro de Lima). We always respect the fronto-ethmoidal wall, which should remain intact.

At this point we may consider the question of the removal of the ethmoid. Woods[12] states in the description of his technique that on the whole a wide removal of the ethmoid is not usually practiced; but if any ethmoidal cells are opened,

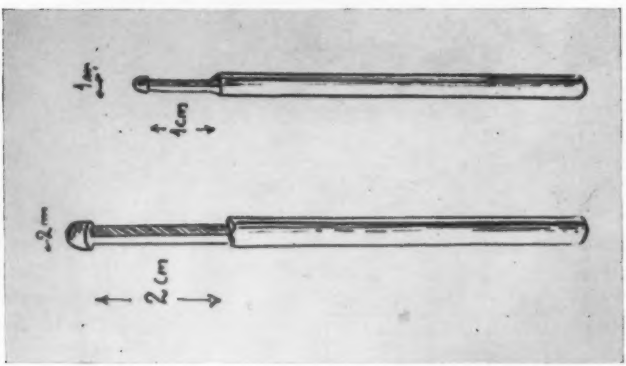

Fig. 5. Burr. Tato's model with smooth spherical head and cutting laterally.

the floor of the frontal sinus is conserved, to avoid making a large opening into the nose, which later would be difficult to close.

He finally adds that his experience does not support the statement that the ethmoid is the key to the frontal sinus.

In this attitude, he has the approval of Mosher[13] who states that in cases of osteomyelitis of the frontal bone and sinus, he often left the infected ethmoidal cells for a further opera-

tion and later found this was not necessary. It would appear that surgeons have been putting the cart before the horse and the logical deduction to be drawn is that the frontal sinus infects the ethmoidal cells, which is contrary to the convictions of many of us.

Fig. 6.

In the presence of mixed pansinusitis (allergy plus infection) characterized by a polypoid degeneration of the ethmoid and maxillary sinuses and to a lesser degree in the sphenoidal, and rarer still, in the frontal, Tato has found that the Ermiro de Lima[14] technique (transmaxillary ethmoido-sphenoidectomy) gives satisfactory results in the great majority of cases.

4. Now comes the original note in the operation, the filling of the cavity with fat or other material. When the osteo-periosteal technique is necessary, in large and high sinuses, the procedure Tato outlines is as follows:

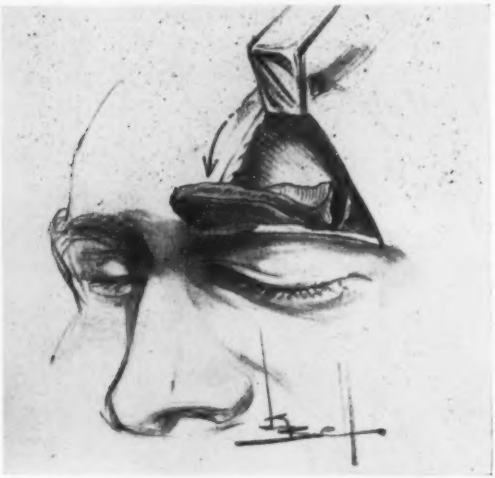

Fig. 7.

a. Radiography of the sinus is made with the addition of a wire grille with square of 1 cm. which is applied to the frontal bone (see Fig. 1) and the head is placed in the fronto-nasal position. Lately we have been following the current practice of a Buenos Aires surgeon, Oliveri, who carefully cuts out the contour of the sinus from the X-ray film, and after the skin has been raised applies it *in situ* over the sinus, so as to enable the surgeon to trace accurately his incision of the periosteum. To achieve the same end, Ermiro de Lima introduces a bronchoscopic lamp into the sinus through a trephine opening. These practices enable one to establish the exact dimensions of the frontal sinus, with a view to future surgery.

b. Anesthesia: Local with pre-anesthesia and, exceptionally, a general anesthetic.

c. Incision of the skin and subcutaneous tissues following the inferior margin of the eyebrow as generally practiced, but extending somewhat laterally (see Fig. 2). The periosteum is not incised. It must be preserved untouched.

d. Separation of the skin and subcutaneous tissue upwards and exposure of the area corresponding to the anter-

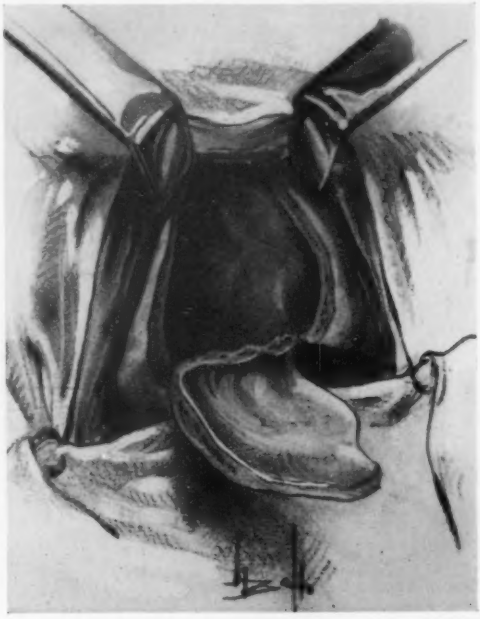

Fig. 8.

ior wall of the sinus with its adherent periosteum and then extending somewhat beyond the sinus limits (see Fig. 3).

e. The periosteum is incised in accordance with the contour of the sinus by studying the grille radiograph (see Fig. 4), with its centimeter squares, or following Oliveri's

or de Lima's methods. By these means a strip of bare bone of 5 mm. width is traced and forms the guide for the future use of the special burr.

f. To enter the sinus please take note of the classical landmark, the nasal process of the frontal bone and perforate it immediately above the lachrymal portion of the os unguis (see Fig. 4). We use a common perforating burr to make the initial step.

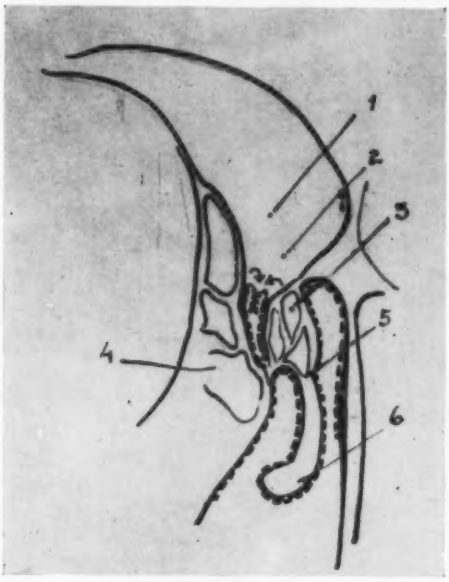

Fig. 9.

1. Frontal sinus, peri-infundibular area.
2. Ostium of the frontal sinus. The arrows indicate the invagination of the peri-infundibular mucous membrane, which is marked by a dotted line.
3. Peri-infundibular cells.
4. Anterior ethmoidal cells.
5. Insertion of middle turbinate.
6. Free portion of middle turbinate.

g. The most useful burr is one especially designed by Tato for frontal sinus work (see Fig. 5). It has three cutting edges and a smooth spherical head, which avoids a

possible perforation of the posterior wall of the sinus and is known as a reamer. It is made by the Storz Instrument Co., of St. Louis. Continuous lavage with normal saline and suction is used during this stage of the operation. The burr is inclined obliquely, so as to obtain a bevelled edge. Starting from the initial perforation, the reamer follows the contour of the sinus, which has previously been established by accurate measurements from the grille radiograph and subsequently traced. The course of the reamer is in and up, and out and down.

h. With a chisel as a lever, the osteoplastic flap is turned downward by fracturing the bone just below the supracil-

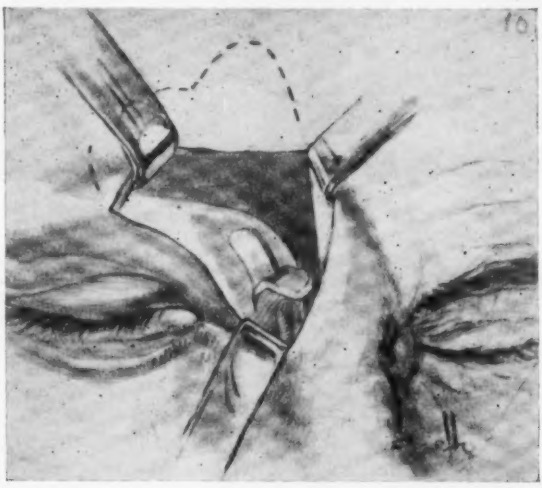

Fig. 10.

iary ridge, the periosteum being left intact. In this way an excellent view of the interior of the sinus is obtained (see Figs. 6, 7, and 8).

i. The mucous membrane of the frontal sinus and that of the interfrontal septum is removed with meticulous care. We use for this purpose a Freer's septal elevator and in

addition a spherical cutting burr or diamond burr of appropriate diameter which is very efficient in narrow hidden recesses. The burr is the best curette. We also eliminate all the intra frontal septa, smoothing them down.

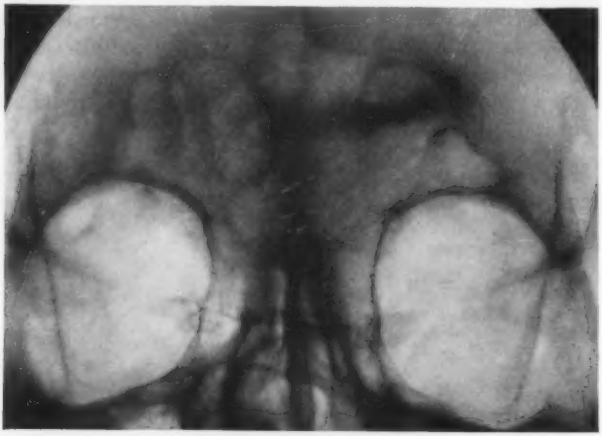

Fig. 11. Pre-operative radiograph.

j. The mucous membrane of the infundibulum is separated from the contour of the duct, invaginated, and pushed downwards to secure permanent closure of the duct (see Fig. 9).

If any doubt exists about a perfect occlusion, a piece of fascia lata attached to the fat is inserted into the opening and left there, or a flap of periosteum from the neighboring bone is inserted as Bergaglio and Alba have done.

k. The cavity is filled with fatty tissue from the abdomen or thigh, and if possible, with the corresponding fibrous tissue sheath attached. We take enough for our purpose. Actually we are now using fibrin sponge, gelatine sponge, coagulated blood, dessicated serum and plasma, with similar results.

l. The osteoperiosteal flap is re-applied and fixed with interrupted sutures.

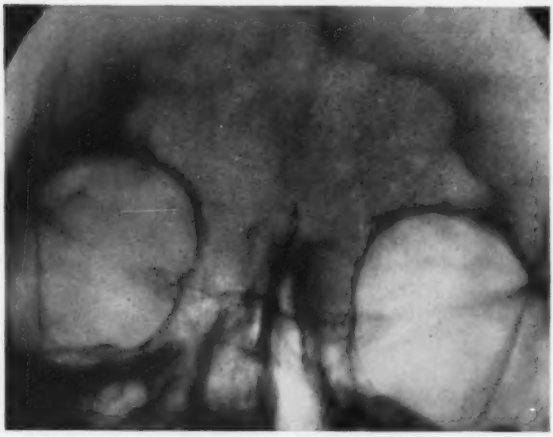

Fig. 12. Post-operative radiograph at 15 days. Note the disappearance of the septa, including intersinal septum, which resection has allowed both sinuses to be operated from one side.

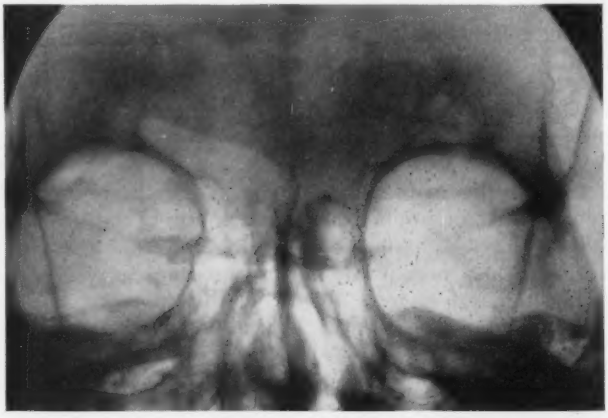

Fig. 13. Post-operative radiograph at 20 months. Note the disappearing contour of the sinuses, formation of new bone and clear space interiorly showing partial reaeration of a cavity.

m. Closure of the skin and the subcutaneous tissue is done separately with interrupted sutures.

n. Application of an elastic bandage to exert a certain degree of pressure. In the cases using this technique the results show healing by first intention. This technique has been employed with good results by Professor Ermiro de Lima of Rio de Janeiro, Professor Moura of Recife, Brazil, Hidalgo of Guayaquil, Ecuador, and by Professor Bergara, Altavista, Sibbald, Romero Diaz of Buenos Aires, and others. Tieffemberg of Buenos Aires prefers inserting bone chips in the cavity and reports success in his two cases.

COMPLICATIONS.

In one of the cases, a tumefaction of moderate degree formed in the supero-internal region of the upper lid which persisted for two weeks and was found to be due to an excess of material placed inside the sinus. It disappeared progressively at the end of a year.

We have followed radiographically various cases and have proved the partial, or almost total, transformation of the bone of the cavity of the sinus, as also a repneumatization or reaeration of limited extent. As this occurred usually in the early cases, we attribute the result as probably due to an insufficient occlusion of the frontal duct (see Figs. 11, 12, and 13).

Recently Tato has operated some acute cases in the presence of complications. Beginning with an ethmoidectomy, he removed the fronto-ethmoidal wall and packed the frontal cavity with gelfoam to which sulpha powder had been added. Postoperatively he applied antibiotic therapy, and eventually the sinus filled with new bone formation. These cases are exceptional, and in no way affect the customary technique described in these pages.

ANALYSIS.

Recently it came to our notice in the "Proceedings of the Fourth International Congress of Otolaryngology" that R. R. Woods of Dublin has carried out a similar operation during the past twenty years. The technique differs in certain de-

tails from Tato's. Woods removes the floor of the sinus in all cases. Before taking away any part of the bone with a forceps he carefully separates off the mucous membrane so that the two are not included in the same bite. This is done to avoid the risk of osteomyelitis.

He removes the mucous membrane of the naso-frontal duct with a successive series of gauze strips. These are pulled down from the sinus through the nose. By gradually increasing their bulk, all the mucous membrane is finally removed, but requiring the use of some force at the end. The use of the curette is forbidden absolutely, because he believes that an infected curette scratching the endosteal surface is another potential cause of osteomyelitis.

Both Tato and Woods stress the complete and absolute removal of all the mucous membrane.

The Woods technique is made more difficult by the incomplete exposure of the high sinus, and it is a tribute to his care and patience, that, working at times blindly, and at others with the help of post-rhinal mirrors, he has been successful in removing every vestige of the mucous membrane and has never left a particle behind to form a cyst or a pocket and so defeat the object of the operation. He has had no case of osteomyelitis.

The exposure of the sinus by the osteoplastic method offers a positive advantage in the discovery of "hideouts" of mucous membrane. Tato feels that the main difference between the two techniques is the insertion of the fat, gelatine sponge, fibrin sponge, etc., to the limit of the capacity of the sinus.

A "dead space" with limited drainage is thus avoided, and if, in addition, the inserted material favors the organization of fibrous tissue by absorbing and holding blood clot, a further claim to its use can be advanced.

Postoperative chemotherapy is observed in both procedures. Woods accepts the inevitable formation of pus in the cavity after the operation, but apparently the cure of the suppuration occurs before the closure of the duct; however,

Tato differs from Woods in claiming that cure by first intention is the rule governing these cases of obliteration of the sinus cavity.

SUMMARY.

1. The occlusion of the frontal sinus cavity with such materials as fat or others is described.

2. The osteoperiosteal flap technique is used in this operation, with an improvement over other similar practices.

3. The treatment of the opposite sinus after resection of the inter-sinal septum can be done without any difficulty.

4. In cases of fracture or fissure of the cribriform plate (ethmoidal fissure or fracture) with cranial hydrorrhea, we are of the opinion that this operation offers the simplest means of approach, for surgical treatment to this area, and it is easier and less dangerous than the intracranial route used by neuro-surgeons.

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THE RECIPIENT OF THE "AWARD OF MERIT" BY THE AMERICAN OTOLOGICAL SOCIETY, INC.

At its annual meeting on May 23, 1954, the American Otological Society, Inc., awarded a Silver Medal, a suitably inscribed scroll and an honorarium of one thousand dollars to Stacy R. Guild, Ph.D., of Baltimore, Maryland. The award was made in recognition of the many contributions made by Dr. Guild in the field of research in otology during the past years.

SPECIAL AWARDS.

At the 75th Annual Meeting of the American Laryngological Association held in Boston, Mass., May 27-28, 1954, Dr. Louis H. Clerf, of Philadelphia, was presented with the gold medal of the deRoaldes Award, and Dr. Henry B. Orton, of Newark, N. J., was presented with the Newcomb Award.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

A joint meeting of the North Carolina Eye, Ear, Nose, and Throat Society and the South Carolina Society of Ophthalmology and Otolaryngology will be held at Durham, N. C., November 4, 5, and 6, 1954. Headquarters will be the Washington Duke Hotel.

Guest otolaryngologists will be Dr. Stanton A. Friedberg of Chicago, Ill., Dr. John Maxwell of Ann Arbor, Michigan, Dr. George Bayling of Durham, N. C. The following guest ophthalmologists will likewise be on the program: Dr. Charles Iliff of Baltimore, Maryland, Dr. John McLean of New York, N. Y., and Dr. Townley Paton of New York, N. Y.

On Wednesday preceding the beginning of the meeting, operative clinics will be held in the various hospitals of Durham and Chapel Hill.

Finally, there will be a football game on Saturday afternoon between the University of North Carolina and the University of South Carolina in Chapel Hill.

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE OF THE AMERICAN MEDICAL ASSOCIATION.

June 1, 1954.

Acousticon Models A-17, A-180 and A-185.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 1, New York.

Auditone Models 11 and 15.
Manufacturer: Audio Co. of America, 5305 N. Sixth St., Phoenix, Ariz.

Audivox Model Super 67 and 70.
Manufacturer: Audivox, Inc., 259 W. 14th St., New York 11, N. Y.

Aurex Models L and M.
Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago, Ill.

Beltone Mono-Pac Model M; Mono-Pac Model "Lyric"; Mono-Pac Model "Rhapsody."
Manufacturer: Beltone Hearing Aid Co., 2900 West 36th St., Chicago 32, Ill.

Cleartone Model 700.
Manufacturer: American Sound Products, Inc., 1303 S. Michigan Ave., Chicago 5, Ill.

Dahlberg Junior Model D-2; Dahlberg Model D-3 Tru-Sonic; Dahlberg Model D-4 Tru-Sonic.
Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.

Fortiphone Models 19-LR; 20A; 21-C and 22.
Manufacturer: Fortiphone Limited, Fortiphone House, 247 Regent St., London W. 1, England.
Distributor: Anton Heilman, 75 Madison Ave., New York 16, N. Y.

Gem Hearing Aid Model V-60.
Manufacturer: Gem Ear Phone Co., Inc., 50 W. 29th St., New York 1, N. Y.

Goldentone Models 25, 69 and 97.
Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40th St., Minneapolis 8, Minn.
Distributor: Goldentone Corp., 708 W. 40th St., Minneapolis 8, Minn.

Maico Model J; Maico Top Secret Model L; Maico Maxitone.
Manufacturer: Maico Co., Inc., 21 North Third St., Minneapolis, Minn.

Micronic Model 303; Micronic Model "Mercury."
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T-10; Microtone Model T-612.

Manufacturer: Microtone Co., Ford Parkway on the Mississippi, St. Paul, Minn.; Minneapolis 9, Minn.

National Ultrathin Model 504; National Vanity Model 506.

Manufacturer: National Hearing Aid Laboratories, 106 So. 7th St., Philadelphia 6, Pa.

Normatone Model C and Model D-53.

Manufacturer: Johnston Hearing Aid Mfg. Co., 708 W. 40 St., Minneapolis, Minn.

Distributor: Normatone Hearing Aid Co., 22 East 7th St., St. Paul (1), Minn.

Otarion Models B-15 and B-30; Otarion Models F-1, and F-3; Otarion Model G-3; Otarion Model H-1; Custom "5."

Manufacturer: Otarion Hearing Aids, 4757 N. Ravenwood, Chicago 40, Ill.

Paravox Model D, "Top-Twin-Tone"; Model J (Tiny Myte).

Manufacturer: Paravox, Inc., 2056 E. 4th St., Cleveland, Ohio.

Radioear Model 62 Starlet; Model 72; Model 82 (Zephyr).

Manufacturer: E. A. Myers & Sons, 306 Beverly Rd., Mt. Lebanon, Pittsburgh, Pa.

Distributor: Radioear Corp., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-16, J-92; Silvertone Model P-15.

Manufacturer: W. E. Johnson Mfg. Co., 708 W. 40th St., Minneapolis, Minn.

Distributor: Sears, Roebuck & Co., 925 S. Homan Ave., Chicago 7, Ill.

Solo-Pak Model 99.

Manufacturer: Solo-Pak Electronics Corp., Linden St., Reading, Mass.

Sonotone Models 910 and 920; Sonotone Model 925; Sonotone Model 940; Sonotone Model 966; Sonotone Model 977; Sonotone Model 988.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

Televox Model E.

Manufacturer: Televox Mfg. Co., 1307 Sansom St., Philadelphia 7, Pa.

Telex Model 99; Telex Model 200; Telex Model 300B; Telex Model 400; Telex Model 500; Telex Model 952; Telex Model 953; Telex Model 1700.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Tonamic Model 50.
Manufacturer: Tonamic, Inc., 12 Russell St., Everett 49, Mass.

Tonemaster; Model Cameo.
Manufacturer: Tonemasters, Inc., 400 S. Washington St., Peoria 2, Ill

Unex Midget Model 95; Unex Midget Model 110; Unex Models 200 and 230.
Manufacturer: Nichols & Clark, Hathorne, Mass.

Vacolite Models J and J-2.
Manufacturer: Vacolite Co., 3003 N. Henderson St., Dallas 6, Tex.

Zenith Miniature 75; Zenith Model Royal; Zenith Model Super Royal; Zenith "Regent."
Manufacturer: Zenith Radio Corp., 6001 Dickens Ave., Chicago, Ill.

All of the accepted hearing devices have vacuum tubes.

Accepted Hearing Aids more than five years old have been omitted from this list for brevity.

TRANSISTOR HEARING AIDS ACCEPTED.

Acousticon Model A300; 1 transistor, 2 tubes. Model A-310; 1 transistor; 2 tubes. Model A-330; 3 transistors. Model A-335; 3 transistors.
Manufacturer: Dictograph Products, Inc., 95-25 149th St., Jamaica 35, New York.

Audivox, Model 71; 3 transistors.
Manufacturer: Audivox, Inc., 123 Worcester St., Boston 18, Mass.

Beltone Concerto Model; 3 transistors.
Manufacturer: Beltone Hearing Aid Co., 2900 W. 36th St., Chicago 32. Illinois.

Maico Transist-Ear, Model O; 3 transistors.
Manufacturer: The Maico Company, Inc., 21 N. 3rd St., Minneapolis 1, Minnesota.

Micronic "All American" Hearing Aid; 3 transistors.
Manufacturer: Audivox, Inc., Successor to Western Electric Hearing Aid Division, 123 Worcester St., Boston 18, Mass.

Microtone Model T1 (Red Dot); 3 transistors. Microtone Model T1 (Yellow Dot); 3 transistors. Microtone Model T31 (Micro-Mite); 1 transistor and 2 tubes.
Manufacturer: The Microtone Corporation, Ford Parkway on the Mississippi, St. Paul 1, Minn.

Otarion Model C-15; 1 transistor, 2 tubes. Otarion Model D-1; 3 transistors. Otarion Model F-22; 1 transistor, 2 tubes.

Manufacturer: Otarion, Inc., 4757 N. Ravenswood Ave., Chicago 40, Ill.

Radioear Model 820; 3 transistors.

Manufacturer: E. A. Myers & Sons, Inc., 306 Beverly Rd., Mt. Lebanon, Pittsburgh 16, Pa.

Silvertone Model H-25; 3 transistors.

Manufacturer: The Dahlberg Co., Golden Valley, Minneapolis 22, Minn.
Distributor: Sears, Roebuck & Co., 925 South Homan Ave., Chicago 7, Illinois.

Sonotone Model 1010; 1 transistor, 2 tubes. Model No. 1111; 3 transistors.

Manufacturer: Sonotone Corporation, Elmsford, N. Y.

Telex Model 954; 1 transistor, 2 tubes. Telex Model 956; 3 transistors.

Manufacturer: Telex, Inc., Telex Park, St. Paul 1, Minn.

Unex Model TR-3D; 3 transistors.

Manufacturer: Nichols & Clark, Hathorne, Mass.

Zenith Model Royal-T; 3 transistors. Zenith Model Super Royal-T; 3 transistors.

Manufacturer: Zenith Radio Corp., 5801 W. Dickens Ave., Chicago 39, Illinois.

SEMI PORTABLE HEARING AIDS.

Ambco Hearing Amplifier (Table Model).

Manufacturer: A. M. Brooks Co., 1222 W. Washington Blvd., Los Angeles 7, Calif.

Aurex (Semi-Portable).

Manufacturer: Aurex Corp., 1117 N. Franklin St., Chicago 10, Ill.

Precision Table Hearing Aid.

Manufacturer: Precision Hearing Aids, 5157 W. Grand Ave., Chicago 39, Ill.

Sonotone Professional Table Set Model 50.

Manufacturer: Sonotone Corp., Elmsford, N. Y.

All of the Accepted hearing devices employ vacuum tubes.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN OTOLOGICAL SOCIETY.

President: Dr. D. E. Staunton Wishart, 170 St. George St., Toronto 5, Ontario, Canada.
Vice-President: Dr. Wm. J. McNally, 1509 Sherbrooke St., West Montreal 25, Canada.
Secretary-Treasurer: Dr. John R. Lindsay, 950 E. 59th St., Chicago 37, Illinois.
Editor-Librarian: Dr. Henry L. Williams, Mayo Clinic, Rochester, Minn.
Meeting: Hollywood Hotel, Hollywood, Fla., March 17-18, 1955.

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Henry B. Orton, Newark, N. J.
First Vice-President: Dr. Jas. H. Maxwell, Ann Arbor, Mich.
Second-Vice-President: Dr. Clyde A. Heatley, Rochester, N. Y.
Secretary: Harry P. Schenck, Philadelphia, Pa.
Treasurer: Fred W. Dixon, Cleveland, Ohio.
Librarian-Historian: Dr. Bernard J. McMahon, St. Louis, Mo.
Meeting: Hollywood Hotel, Hollywood, Fla., March 13-14, 1955.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Kenneth M. Day, 121 University Pl., Pittsburgh, Pa.
President-Elect. Dr. Dean M. Lierle, Iowa City, Iowa.
Secretary: Dr. C. Stewart Nash, 277 Alexander St., Rochester, N. Y.
Meeting: Hollywood Hotel, Hollywood, Fla., March, 1955.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Sam Sanders, Memphis, Tenn.
Vice-Chairman: Dr. Alden H. Miller, Los Angeles, Calif.
Secretary: Dr. Hugh A. Kuhn, Hammond, Ind.
Meeting: San Francisco, Calif., June 21-25, 1954.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Walter H. Theobald, 307 N. Michigan Ave., Chicago 11, Ill.
President-Elect: Dr. Algernon B. Reese, 73 East 71st St., New York 21, N. Y.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Waldorf-Astoria, New York City, Sept. 19-24, 1954.

AMERICAN BOARD OF OTOLARYNGOLOGY.

Meeting: Hotel Marshall, Richmond, Va., March 6-10, 1955.
Waldorf-Astoria, New York City, Sept. 13-17, 1954.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, 1100 No. Charles St., Baltimore 1, Md.
Secretary: Dr. F. Johnson Putney, 255 So. 17th St., Philadelphia (3) Pa.
Meeting:

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Clifton E. Benson, Bremerton, Wash.
President-Elect: Dr. Carl D. F. Jensen, Seattle, Wash.
Secretary: Dr. Willard F. Goff, 1215 Fourth Ave., Seattle, Wash.

THE SECTION OF OTOLARYNGOLOGY OF THE MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA.

Chairman: Dr. John Louzan.
Vice-Chairman: Dr. Willard B. Walters.
Secretary: Dr. Jack L. Levine.
Treasurer: Dr. Russell S. Page, Jr.
Meetings are held on the third Tuesday of October, November, March and May, 7:00 P.M.
Place: Army and Navy Club, Washington, D. C.

THE LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. W. L. Hughes, Lamar Life Bldg., Jackson, Miss.
Vice-President: Dr. Ralph H. Riggs, 1513 Line Ave., Shreveport, La.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.

OTOSCLEROSIS STUDY GROUP.

President: Theo. E. Walsh, 640 So. Kingshighway, St. Louis 10, Mo.
Secretary: Dr. Lawrence R. Boies, Med. Arts Bldg., Minneapolis 2, Minn.
Meeting: Waldorf-Astoria, New York City, Sept., 1954.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Albert D. Ruedemann, 1633 David Whitney Bldg., Detroit 26, Mich.
President-Elect: Dr. F. Lambert McGannon, 14900 Detroit Ave., Lakewood 9, Ohio.
Secretary-Treasurer: Dr. Michael H. Barone, 468 Delaware Ave., Buffalo 2, N. Y.
Meeting: Waldorf-Astoria, New York City, September, 1954.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. J. M. Tato, Azcuenaga 235, Buenos Aires, Argentina.
Executive Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa., U. S. A.
Meeting: Fifth Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
President: Dr. J. H. Font, Medical Arts Bldg., San Juan, P. R.
Time and Place: 1956, Puerto Rico.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Norris J. Heckel, Chicago, Ill.
President-Elect: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.
Meeting: Chicago, Ill., Sept. 22-24, 1954.

THE VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Peter N Pastore, Richmond, Va.
President-Elect: Dr. G. S. Fitz-Hugh, Charlottesville, Va.
Vice-President: Dr. H. L. Mitchell, Lexington, Va.
Secretary-Treasurer: Dr. L. B. Sheppard, 301 Medical Arts Bldg., Richmond, Va.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Harold Owens, M.D.
Secretary-Treasurer: Robert A. Norene, M.D.
Chairman of Section on Ophthalmology: Sol Rome, M.D.
Secretary of Section on Ophthalmology: Wendell C. Irvine, M.D.
Chairman of Section on Otolaryngology: Max E. Pohlman, M.D.
Secretary of Section on Otolaryngology: Herschel H. Burston, M.D.
Place: Los Angeles County Medical Association Building, 1925 Wilshire Boulevard, Los Angeles 57, Calif.
Time: 6:00 P.M., first Thursday of each month from September to June inclusive—Ophthalmology Section. 6:00 P.M., fourth Monday of each month from September to June inclusive—Otolaryngology Section.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Harry Nievert, 555 Park Ave., New York (21), N. Y.
Secretary: Dr. Louis Joel Fleit, 66 Park Ave., New York (16), N. Y.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Cecil Swann, Asheville, N. C.
Secretary and Treasurer: Dr. Geo. B. Ferguson, Durham, N. Car.
Meeting: Joint, with South Carolina Society of Ophthalmology and Otolaryngology, Durham, N. C., Nov. 4-6, 1954.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. David S. Asbill, Columbia, S. Car.
Vice-President: Dr. John McLean, Greenville, S. Car.
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. Car.
Meeting: Joint, with North Carolina Eye, Ear, Nose and Throat Society, Durham, N. C., Nov. 4-6, 1954.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Secretary-Treasurer: Dr. John F. Tolan, 3419 47th Ave., Seattle (5), Wash.
Meeting: Honolulu, 1954.

THE RESEARCH STUDY CLUB OF LOS ANGELES, INC.

Chairman: Dr. Isaac H. Jones, 635 S. Westlake, Los Angeles, Calif.
Treasurer: Dr. Pierre Violé, 1930 Wilshire Blvd., Los Angeles, Calif.
Program Chairmen:
Otolaryngology: Dr. Leland G. Hunnicutt, 98 N. Madison Ave., Pasadena, Calif.
Ophthalmology: Dr. Harold F. Whalman, 727 W. 7th St., Los Angeles, Calif.
Mid-Winter Clinical Convention annually the last two weeks in January at Los Angeles, Calif.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Chas. C. Grace, 145 King St., St. Augustine, Fla.
President-Elect: Dr. Jos. W. Taylor, 706 Franklin St., Tampa, Fla.
Secretary-Treasurer: Dr. Carl S. McLemore, 1217 Kuhl Ave., Orlando, Fla.

THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

President: Dr. Harry P. Schenck.
Vice-President: Dr. William J. Hitschler.
Treasurer: Dr. Chevalier L. Jackson.
Secretary: Dr. John J. O'Keefe.
Historian: Dr. Herman B. Cohen.
Executive Committee: Dr. M. Valentine Miller, Dr. Charles E. Towson, Dr. Thomas F. Furlong, Dr. Benjamin H. Shuster, ex-officio.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Vice-Chairman: Dr. K. W. Cosgrove, 111 E. Capitol Ave., Little Rock, Ark.
Secretary: Dr. F. A. Holden, Medical Arts Bldg., Baltimore, Md.
Meeting:

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. James K. Stewart, Wheeling, W. Va.
Secretary-Treasurer: Dr. Frederick C. Reel, Charleston, W. Va.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. C. Otrich, Belleville, Ill.
President-Elect: Dr. Phil R. McGrath, Peoria, Ill.
Secretary-Treasurer: Dr. Alfred G. Schultz, Jacksonville, Ill.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Oscar Marchman, Jr., Dallas, Texas.
Secretary-Treasurer: Dr. Morris F. Waldman, Dallas, Texas.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE

President: Dr. Robert Black, 401 Medical Arts Bldg., Winnipeg, Manitoba.
Secretary: Dr. W. Ross Wright, 361 Regent St., Fredericton, N. B.
Place: Harrison Hot Springs Spa, Harrison Hot Springs, B. C.
Time: June 13-15, 1954.

BALTIMORE NOSE AND THROAT SOCIETY

Chairman: Dr. Albert Steiner, 1308 Eutaw Pl., Baltimore, Md.
Secretary-Treasurer: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Maryland.

SOCIEDAD DE OTO-RINO-LARINGOLOGIA, COLEGIO MEDICO DE EL SALVADOR, SAN SALVADOR, C. A.

President: Dr. Salvador Mixco Pinto.
Secretary: Dr. Daniel Alfredo Alfaro.
Treasurer: Dr. Antonio Pineda M.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzalez Ulloa, Mexico, D. F.
Secretary: Dr. Juan de Dios Peza, Mexico, D. F.

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Sub-Secretario del Exterior: Dr. Oreste E. Bergaglio.
Secretario del Interior: Dr. Eduardo Casterán.
Sub-Secretario del Interior: Dr.Atilio Viale del Carril.
Secretario Tesorero: Dr. Vicente Carri.
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ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presdente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perelló, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

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Presidente: Dr. Reinaldo de Villiers.
Vicepresidente: Dr. César Cabrera Calderin.
Secretario: Dr. José Xirau.
Tesorero: Dr. Alfredo M. Petit.
Vocal: Dr. José Gross.
Vocal: Dr. Pedro Hernández Gonzalo.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Andre Soulas, Paris, France.
Secretary: Dr. Chevalier L. Jackson, 1901 Walnut St., Philadelphia 3, Pa. U. S. A.
Meeting: 3rd International Congress of Broncho-Esophagology.
Time and Place: September or October, 1954, Lisbon, Portugal.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER — CAMPINAS.

President: Dr. Heitor Nascimento.
First Secretary: Dr. Roberto Barbosa.
Second Secretary: Dr. Roberto Franco do Amaral.
Librarian-Treasurer: Dr. Leoncio de Souza Queiroz.
Editors for the Archives of the Society: Dr. Guedes de Melo Filho, Dr. Penido Burnier and Dr. Gabriel Porto.

SOCIEDAD CUBANA DE OTO-LARINGOLOGIA.

President: Dr. Reinaldo de Villiers.
Vice-President: Dr. Jorge de Cárdenas.
Secretary: Dr. Pablo Hernandez.

SOCIEDAD DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOSCOPIA DE CORDOBA.

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Vice-Presidente: Dr. Luis E. Olsen.
Secretario: Dr. Eugenio Romero Díaz.
Tesorero: Dr. Juan Manuel Pradales.
Vocales: Dr. Osvaldo Suárez, Dr. Nondier Asís R., Dr. Jorge Bergallo Yofre.

BUENOS AIRES CLUB OTORINOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTORRINOLARINGOLOGIA (BOGOTA, COLOMBIA).

Presidente: Dr. Alfonso Tribin P.
Secretario: Dr. Felix E. Lozano.
Tesorero: Dr. Mario Arenas A.

SOCIEDAD ESPANOLA DE OTORRINOLARINGOLOGIA.

Presidente: Dr. D. Adolfo Hinojar Pons.
Vice-Presidente: Dr. D. Jose Perez Mateos.
Secretario General: Dr. D. Francisco Marañés.
Tesorero: Dr. D. Ernesto Alonso Ferrer.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

SOCIEDAD DE ESTUDIOS CLINICOS DE LA HABANA

Presidente: Dr. Frank Canosa Lorenzo.
Vice-Presidente: Dr. Julio Sanguily.
Secretario: Dr. Juan Portuondo de Castro.
Tesorero: Dr. Luis Ortega Verdes.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting: Lima, Peru, 1957.

SIXTH INTERNATIONAL CONGRESS OF OTOLARYNGOLOGY

President: Dr. Arthur W. Proetz, Beaumont Bldg., St. Louis, Mo.
General Secretary: Dr. Paul Holinger, 700 No. Michigan Ave., Chicago (11), Ill.
Meeting: U. S. A., 1957.

SOCIEDADE PORTUGUESA DE OTORRINOLARINGOLOGIA E DE BRONCO-ESOFAGOLOGIA

Presidente: Dr. Alberto Luis de Mendonca.
Vice-Presidente: Dr. Jaime de Magalhaes.
1.° Secretario: Dr. Antonio da Costa Quinta.
2.° Secretario: Dr. Albano Coelho.
Tesoureiro: Dr. Jose Antonio de Campos Henriques.
Vogais: Dr. Teofilo Esquivel.
Dr. Antonio Cancela de Amorim.
Sede: Avenida da Liberdade, 65, 1.°, Lisboa.

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